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Development of a Paradigm for Examining Social Exclusion in Schizophrenia: Dissociating Expectancy Violations from Social Distress

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Development of a Paradigm for Examining Social Exclusion in Schizophrenia: Dissociating Expectancy Violations from Social Distress

A dissertation proposal submitted in partial satisfaction of the
requirements for the degree of a Doctor of Philosophy
in Psychology

by

Peter Eugene Clayson

2017

ABSTRACT OF THE DISSERTATION

Development of a Paradigm for Examining Social Exclusion in Schizophrenia: Dissociating Expectancy Violations from Social Distress

by

Peter Eugene Clayson

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2017

Professor Gregory Allen Miller, Co-Chair

Professor Cindy M. Yee-Bradbury, Co-Chair

Despite the well-established finding that psychosocial stressors influence symptom onset and clinical relapse in patients with schizophrenia, less is known about how these stressors influence such changes in schizophrenia and why only a subset of patients relapse. Stress elicited by social evaluative threat (SET) may provide a mechanism by which psychosocial stressors influence symptoms and relapse. The present study sought to develop a modified Cyberball paradigm to examine social exclusion, a type of SET, in patients in an effort to understand how sensitivity to SET impacts symptoms and relapse. Additionally, there is controversy regarding the functional significance of the activation of the dorsal anterior cingulate cortex (dACC) region of the brain during the Cyberball task, with some studies implicating dACC in detecting violations of expectancy and other studies implicating dACC in responding to social distress and negative affect. The present examination evaluated the functional significance of exclusion-related neural activity during the task. Electroencephalogram (EEG) data were recorded while 32 healthy participants completed a modified Cyberball paradigm. EEG data were fit to a source model with six regional sources, including dACC and anterior insula (AI). Participants self-reported similar levels of social distress to those reported in prior Cyberball studies, suggesting that the social exclusion manipulation was successful. Present findings provided evidence that early dACC activity is sensitive to violations of expectancy and that late dACC activity is sensitive to changes in negative affect. Bilateral AI activity was greater during the first block of the exclusion phase than during the inclusion phase and positively correlated with measures of social distress, supporting previous research implicating AI in responding to changes in social distress following exclusion. By identifying the functional significance of scalp- and source-space activity during a modified Cyberball task, these findings lay the groundwork for examining SET in patients with schizophrenia. This study capitalized on the temporal resolution of EEG in order to identify the functional significance of early and late dACC activity and provided evidence that the functional significance of dACC is not fully accounted for by either expectancy violations or social distress and negative affect alone.

The dissertation of Peter Eugene Clayson is approved.

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2017

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Abbreviated Curriculum Vitae

Education

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Selected Publications

Clayson, P. E., & Miller, G. A. (2017). ERP Reliability Analysis (ERA) Toolbox: An open-source toolbox for analyzing the reliability of event-related brain potentials. *International Journal of Psychophysiology, 111,* 68-79. doi: 10.1016/j.ijpsycho.2016.10.012

Clayson, P. E., & Miller, G. A. (2017). Psychometric considerations in the measurement of event-related brain potentials: Guidelines for measurement and reporting. *International Journal of Psychophysiology, 111,* 57-67. doi: 10.1016/j.ijpsycho.2016.09.005

Larson, M. J., **Clayson, P. E.,** Kirwan, C. B., & Weissman, D. H. (2016). Event-related potential indices of congruency sequence effects without feature integration or contingency learning confounds. *Psychophysiology*, *53*, 814-822. doi: 10.1111/psyp.12625

Baldwin, S. A., Larson, M. J., & **Clayson, P. E.** (2015). The dependability of electrophysiological measurements of performance monitoring in a clinical sample: A generalizability and decision analysis of the ERN and Pe. *Psychophysiology*, *52*, 790-800. Reprinted in virtual issue of *Psychophysiology*: Error-related negativity (ERN) (2016).

Larson, M. J.[†], **Clayson, P. E.**[†], Primosch, M., Leyton, M., & Steffensen, S. C. (2015). The effects of acute dopamine precursor depletion on the cognitive control functions of performance monitoring and conflict processing: An event-related potential (ERP) study. *PLoS ONE*, *10*, e0140770. doi: 10.1371/journal.pone.0140770

Baldwin, S. A., Larson, M. J., & **Clayson, P. E.** (2015). The dependability of electrophysiological measurements of performance monitoring in a clinical sample: A generalizability and decision analysis of the ERN and Pe. *Psychophysiology*, *52*, 790-800. doi: 10.1111/psyp.12401

Clawson, A., **Clayson, P. E.**, South, M., Bigler, E. D., & Larson, M. J. (2015). An electrophysiological investigation of interhemispheric transfer time in individuals with high-functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *45*, 363-375. doi: 10.1007/s10803-013-1895-7

Miller, G. A., **Clayson, P. E.**, & Yee, C. M. (2014). Hunting genes, hunting endophenotypes. *Psychophysiology*, *51*, 1329-1330. doi: 10.1111/psyp.12354

Larson, M. J.[†], **Clayson, P. E.**[†], & Clawson, A.[†] (2014). Making sense of all the conflict: A theoretical review and critique of conflict-related ERPs. *International Journal of Psychophysiology*, *93*, 283-297. doi: 10.1016/j.ijpsycho.2014.06.007

Clawson, A., **Clayson, P. E.**, Worsham, W., Johnston, O., South, M., & Larson, M. J. (2014). How about watching others? Observation of error-related feedback by others in autism spectrum disorders. *International Journal of Psychophysiology*, *92*, 26-24. doi: 10.1016/j.ijpsycho.2014.01.009

Clayson, P. E., & Larson, M. J. (2013). Psychometric properties of conflict monitoring and conflict adaptation indices: Response time and conflict N2 event-related potentials. *Psychophysiology*, *50*, 1209-1219. doi: 10.1111/psyp.12138

Clayson, P. E., & Larson, M. J. (2013). Adaptation to emotional conflict: Evidence from a novel paradigm. *PLoS ONE*, *8*, e76776. doi: 10.1371/journal.pone.0075776

Larson, M. J., Clawson, A., **Clayson, P. E.**, & Baldwin, S. A. (2013). Cognitive conflict adaptation in generalized anxiety disorder. *Biological Psychology*, *94*, 408-418. doi: 10.1016/j.biopsycho.2013.08.006

Clawson, A., **Clayson, P. E.**, & Larson, M. J. (2013). Cognitive control adjustments and conflict adaptation in major depressive disorder. *Psychophysiology*, *50*, 711-721. doi: 10.1111/psyp.12066

Clayson, P. E., Baldwin, S. A., & Larson, M. J. (2013). How does noise affect amplitude and latency measurement of event-related potentials (ERPs)? A methodological critique and simulation study. *Psychophysiology*, *50*, 174-186. doi: 10.1111/psyp.12001

Clayson, P. E., Clawson, A., & Larson, M. J. (2012). The effects of induced state negative affect on performance monitoring processes. *Social, Cognitive, and Affective Neuroscience*, *7*, 677-688. doi: 10.1093/scan/nsr040

Clayson, P. E., & Larson, M.J. (2012). Cognitive performance and electrophysiological indices of cognitive control: A validation study of conflict adaptation. *Psychophysiology*, *50*, 426-433. doi: 10.1111/j.1469-8986.2011.01345.x

Larson, M. J., **Clayson, P. E.**, & Farrer, T. J. (2012). Performance monitoring and cognitive control in individuals with mild traumatic brain injury. *Journal of the International Neuropsychological Society*, *18*, 323-333. doi: 10.1017/S1355617711001779

Larson, M. J., **Clayson, P. E.**, & Baldwin, S. A. (2012). Performance monitoring following conflict: Internal adjustments in cognitive control. *Neuropsychologia*, *50*, 426-433. doi: 10.1016/j.neuropsychologia.2011.12.021

Clayson, P. E., & Larson, M. J. (2011). Effects of repetition priming on neurophysiological and behavioral indices of conflict adaptation and cognitive control. *Psychophysiology*, *48*, 1621-1630. doi: 10.1111/j.1469-8986.2011.01265.x

Larson, M. J., Farrer, T. J., & **Clayson, P. E.** (2011). Cognitive control in mild traumatic brain injury: Conflict monitoring and conflict adaptation. *International Journal of Psychophysiology*, *82*, 69-78. doi: 10.1016/j.ijpsycho.2011.02.018

Clayson, P. E., & Larson, M. J. (2011). Conflict adaptation and sequential trial effects: Support for the conflict monitoring theory. *Neuropsychologia*, *49*, 1953-1961. doi: 10.1016/j.neuropsychologia.2011.03.023

Clayson, P. E., Clawson, A., & Larson, M. J. (2011). Sex differences in electrophysiological indices of conflict monitoring. *Biological Psychology*, *87*, 282-289. doi: 10.1016/j.biopsycho.2011.03.011

Larson, M. J., & **Clayson, P. E** (2011). The relationship between cognitive performance and electrophysiological indices of performance monitoring. *Cognitive, Affective, and Behavioral Neuroscience*, *11*, 159-171. doi: 10.3758/s13415-010-0018-6

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Introduction

Prevailing theoretical explanations of symptom onset and clinical relapse in patients with schizophrenia emphasize the role of psychosocial stressors (Horan et al., 2005; Meyer-Lindenberg & Tost, 2012; Morgan, Charalambides, Hutchinson, & Murray, 2010; Nuechterlein et al., 1994; Phillips, Francey, Edwards, & McMurray, 2007; Walker, Mittal, & Tessner, 2008). Potent features of psychosocial stressors include social evaluative threat (SET). Specifically, SET is a salient stressor that involves an instance when the self-identity is, or could be, negatively judged, such as during critical or hostile interpersonal interactions (Dickerson & Kemeny, 2004). There is evidence to suggest that patients with psychosis report experiencing more stress overall in interpersonal interactions than healthy individuals (Myin-Germeys, Van Os, Schwartz, Stone, & Delespaul, 2001). Moreover, Sholty (2012) observed that stressors characterized by SET over the previous six months as well as chronic interpersonal stress predicted overall positive and negative symptoms in patients with schizophrenia. Patients with schizophrenia experience various psychosocial stressors related to SET, including stigmatization and environments characterized by high expressed emotion (EE), that impact symptoms and clinical relapse.

Stigma-related stress occurs when a patient views a stigma-related stressor as harmful to the patient’s self-identity and as exceeding the patient’s skills to cope (Major & O’Brien, 2005). In patients with schizophrenia, stigma-related stress is related to increased positive symptoms (e.g., Gerlinger et al., 2013). However, mixed findings have been observed for the relationship between stigma-related stress and symptom onset and clinical relapse (Gerlinger et al., 2013; Rüsch et al., 2014; Świtaj, Wciórka, Smolarska-Świtaj, & Grygiel, 2009). Despite findings of stigma-related stress as a predictor of positive symptoms, less is known about how stigma influences such changes in schizophrenia and why only a subset of patients relapse.

When patients diagnosed with schizophrenia reside in family and residential environments characterized by high EE, these individuals have a greater risk for relapse than patients living in low-EE environments (for meta-analysis, see Butzlaff & Hooley, 1998). Relationships characterized by high EE often reflect problematic levels of emotional over-involvement, hostility, and criticism that purportedly drive transactional patterns and the emotional climate of the environment (Brown, Birley, & Wing, 1972; Hooley, 2007). This association has been established in a number of studies utilizing various approaches to measure EE, whether scoring speech patterns in interviews with relatives or assessing patients' perceptions of criticism (e.g., Baker, Kazarian, Helmes, Ruckman, & Tower, 1987; Lebell et al., 1993; Tompson et al., 1995). Prevailing models of the association between EE and relapse emphasize the role of a high-EE environment as a stressor for patients (Brown et al., 1972; Cutting, Aakre, & Docherty, 2006; Kuipers et al., 2006; Nuechterlein & Dawson, 1984). Potent features of high-EE environments, such as problematic levels of hostility and criticism, represent interpersonal interactions characterized by SET.

Stress elicited by SET may provide a mechanism by which psychosocial stressors, such as stigmatization and high-EE environments, influence psychiatric symptoms and relapse in schizophrenia. For example, patients who display greater sensitivity to SET may show a stronger relationship between psychosocial stressors and symptom exacerbation, which could account for why only some patients experience stress-related relapse. Thus, identifying and evaluating individual differences in sensitivity to SET may serve to highlight a key characteristic of those patients who are most vulnerable to psychosocial stressors and thus at greatest risk for increased symptom severity and relapse. Furthermore, explicating the mechanism by which psychosocial stressors influence the exacerbation of symptoms and relapse will contribute towards advancing theories about how interpersonal stressors culminate in negative clinical outcomes. And while the proposed study targets developing a paradigm for examining SET in patients with schizophrenia, psychosocial stressors impact symptoms in other clinical populations. For example, in numerous clinical disorders stigma-related stressors have been shown to be associated with psychiatric symptoms and reduced treatment adherence (for reviews, see Alonso et al., 2009; Livingston & Boyd, 2010) and high EE has been shown to predict clinical relapse (for review, see Hooley, 2007). Therefore, identifying potential candidates for the mechanism linking psychosocial stressors to clinical relapse has the potential to cut across diagnostic categories and to be applied to understanding how psychosocial stressors influence symptom onset and relapse in other clinical disorders.

Stress and Schizophrenia

Research examining the relationship between stress, symptom onset, and symptom expression or change in schizophrenia has often focused on significant life stressors that are uncontrollable and independent of the illness, such as loss of a family member, trauma, and parental divorce (e.g., Nuechterlein & Dawson, 1984; Nuechterlein et al., 1994; Phillips et al., 2007; Ventura, Nuechterlein, Lukoff, & Hardesty, 1989). Results of cross-sectional studies investigating whether patients with schizophrenia experience more significant life stressors than do healthy controls are somewhat inconsistent (for reviews, see Phillips et al., 2007; Walker et al., 2008), but results obtained from longitudinal studies do indicate an increase in the number of life stressors prior to psychosis relapse (e.g., Lataster, Myin-Germeys, Lieb, Wittchen, & Van Os, 2012; Mondelli et al., 2008; Nuechterlein et al., 1994; Ventura et al., 1989).

These studies have tended to focus on the number of life stressors experienced, rather than on a patient's interpretation of the event as stressful. A patient's appraisal of a stressor as uncontrollable, unmanageable, and undesirable may be more important than whether the event would be considered a stressor in a normative population, as schizophrenia patients may demonstrate heightened sensitivity to stressors relative to healthy controls that potentially leads to higher stress (Horan et al., 2005; Renwick et al., 2009). Schizophrenia patients, for example, have reported life stressors as less controllable and more poorly managed than healthy individuals despite patients reporting fewer life stressors (Horan et al., 2005). Patients with schizophrenia also report higher levels of distress to daily life stressors than do controls and are more likely to report distress to seemingly innocuous events (Beck & Worthen, 1972; Norman & Malla, 1991, 1993). In addition to heightened reactivity to daily stressors (Docherty, St-Hilaire, Aakre, & Seghers, 2009), self-reported stress in response to daily stressors is positively correlated with symptoms of psychosis, depression, and anxiety (for review, see Phillips et al., 2007). In a study using experience-sampling methodology, patients with a psychotic disorder, their first-degree relatives, and healthy controls reported the number of daily stressors and their emotional reaction to those stressors (Myin-Germeys et al., 2001). Patients reported experiencing more stressors than did first-degree relatives and controls and greater increases with negative affect and greater decreases in positive affect to those stressors than relatives or controls. Notably, the stressors experienced by patients tended to be more social in nature than were the stressors reported by relatives or controls. The notion that social stressors may be more potent for patients than healthy individuals is consistent with research identifying social environmental risk factors for symptom onset and exacerbation in schizophrenia.

Indeed, there is substantial evidence indicating increased rates of psychosis in the context of risk factors that are social in nature (for review, see van Os, Kenis, & Rutten, 2010). For example, minority group status is associated with increased rates of psychosis in first-generation migrants that persists in second-generation migrants (for meta-analyses, see Bourque, van der Ven, & Malla, 2011; Cantor-Graae & Selten, 2005; Kirkbride et al., 2012; Selten, Cantor-Graae, & Kahn, 2007) as well as in minority groups without recent migration (Bresnahan et al., 2007). Risk is reduced when the minority group occupies a larger proportion of a neighborhood than smaller minority groups, suggesting that minority position rather than migrant status is a critical factor contributing to psychosis onset (Morgan et al., 2010; Veling et al., 2008). Other meta-analytic work indicates a dose-response relationship between psychotic symptoms and urbanicity, which is variably defined but often refers to residence in large urban areas (for meta-analyses, see Kelly et al., 2010; Krabbendam & van Os, 2005; March et al., 2008; Vassos, Pedersen, Murray, Collier, & Lewis, 2012). Indeed, moving from urban to rural areas during childhood appears to decrease the risk for psychosis, suggesting factors specific to urban environments are contributing to increased risk (Pedersen & Mortensen, 2001). Because minority group status and urbanicity are environmental risk factors that may lead to increased exposure to daily stressors related to social disadvantage, exclusion, or defeat, increased chronic levels of stress appears to be a key influence (Hoffman, 2007; Morgan et al., 2008; Selten & Cantor-Graae, 2005; Selten, van der Ven, Rutten, & Cantor-Graae, 2013; van Os et al., 2010).

A circumstance that may mediate the relationship between social stressors and daily levels of stress is social support. Patients with schizophrenia often have smaller social networks that are more interconnected and provide less support than do those of healthy controls (Bengtsson-Tops & Hansson, 2001; Cohen & Sokolovsky, 1978; Hammer, 1963, 1981; Patterson et al., 1997;

Semple et al., 1997; Sokolovsky, Cohen, Berger, & Geiger, 1978; Tolsdorf, 1976). Lower levels of social support are associated with more positive and negative symptoms (Bengtsson-Tops & Hansson, 2001) and higher rates of re-hospitalization (Bergen, Hunt, Armitage, & Bashir, 1998). Alternatively, higher social support may serve as a protective factor that facilitates coping and competence and reduces deleterious effects of daily stressors (Buchanan, 1995). Other evidence for social support serving as a protective factor comes from studies of psychosis indicating that higher levels of social support are associated with better functional outcome (Howard, Leese, & Thornicroft, 2000), higher life satisfaction (Koivumaa-Honkanen, Honkanen, Antikainen, Hintikka, & Viinamäki, 1999), and lower levels of positive symptoms and fewer hospitalizations over a three-year period (Norman et al., 2005). Given the relationship between social support and various outcomes in schizophrenia, levels of social support may exacerbate or buffer the deleterious effects of stress, as has been proposed for other psychiatric disorders, such as depression (Ibarra-Rovillard & Kuiper, 2011).

Summary

Schizophrenia patients' heightened reactivity to daily stressors and exposure to environmental risk factors may worsen the impact of those stressors. Compared to healthy individuals, it appears that patients demonstrate not only greater vulnerability to daily stressors but they may be especially sensitive to stress that is social in nature. Given this vulnerability, sensitivity to social stress may represent a critical susceptibility factor that increases risk for more severe clinical symptoms and relapse in schizophrenia.

Social Exclusion

Forming and maintaining social connections is a common motivation in human beings (Baumeister & Leary, 1995). The strength of these social connections has been related to positive outcomes, such as physical (Eisenberger, 2013; Eisenberger & Cole, 2012; Seeman, 1996) and mental health (Smith, Murphy, & Coats, 1999). Indeed, in healthy samples social isolation contributes to poor overall cognitive function, rapid cognitive decline, high sensitivity to social threats, and morbidity (Cacioppo & Hawkley, 2009). The loneliness that comes from social isolation can result in social pain (Cacioppo & Hawkley, 2009), which refers to the painful feelings elicited by social loss, rejection, or exclusion (Eisenberger & Lieberman, 2004; Macdonald & Leary, 2005). From an evolutionary perspective, social pain may serve a function similar to that of physical pain; insofar as physical pain focuses attention, social pain may focus attention on significant events in order to flexibly adjust behavior to adapt to future social interactions and avoid further exclusion (Cacioppo & Hawkley, 2009; Macdonald & Leary, 2005; Thornhill & Thornhill, 1989).

Physical-Social Pain Overlap

Pharmacological, neurological, and neuroimaging work on the relationship between brain systems related to physical pain and those associated with social pain indicates considerable overlap between these systems (Eisenberger, 2012, 2015). The experience of pain can be divided into two separate constructs—pain sensation, referring to nociceptive information, and pain affect (Rainville, 2002). Pain affect refers to unpleasant feelings associated with pain sensation. Pain affect motivates adaptive behavior to avoid further pain and, in social situations, motivates adaptive behavior to warn against potential social harm (Price, 2000).

Two brain areas associated with pain affect in response to physical pain are dorsal anterior cingulate cortex (dACC) and anterior insula (AI; Price, 2000; Treede, Kenshalo, Gracely, & Jones, 1999). For example, dACC or AI lesions dull the unpleasant affective component of physical pain while sparing the sensory component of pain (Berthier, Starkstein, & Leiguarda, 1988; Foltz & White Jr, 1962). Self-report measures of the unpleasant affective component of pain are also correlated with dACC and AI activity (Craig, 2002; Craig, Reiman, Evans, & Bushnell, 1996; Kulkarni et al., 2005; Tolle et al., 2001), whereas pain sensation, based on self-report measures of pain intensity, appears to be associated with activation in the somatosensory cortex and posterior insula (for review, see Peyron, Laurent, & García-Larrea, 2000). Both dACC and AI have also been implicated in response to social pain, suggesting these brain regions as points of overlap between brain systems activated in response to physical and social pain (Eisenberger, 2012, 2015).

Some evidence for the role of ACC in pain affect associated with social interactions comes from studies of separation distress behaviors and social interactions in animals. Following separation from their mothers, young mammals displayed fewer behaviors aimed at reducing mother-infant separation when ACC was lesioned (Hadland, Rushworth, Gaffan, & Passingham, 2003; MacLean & Newman, 1988). Conversely, ablation of cingulate gyrus in rodent mothers leads to disrupted maternal responses and reduced maternal behavior, despite rodent mothers displaying other species-typical behavior (Murphy, MacLean, & Hamilton, 1981), although results of this study apply to the role of cingulate gyrus broadly rather than to ACC specifically. Furthermore, ACC lesions in monkeys lead to fewer social interactions and less time spent in proximity to other monkeys (Hadland et al., 2003). Limitations of the generalizability of animal research to human behavior notwithstanding, these studies provide support for the notion that ACC is involved in social pain in humans by evidencing the effects of ACC lesions on social interaction behaviors in animals.

Social exclusion appears to elicit pain affect, similar to the affective component of physical pain (Eisenberger, 2012, 2015; Eisenberger & Lieberman, 2004; Macdonald & Leary, 2005).

Social exclusion refers to the rejection or separation from others that involves the threat or loss of an interpersonal relationship (Macdonald & Leary, 2005). Social exclusion is a type of SET, as exclusion from a group represents possible negative judgment from group members and thus risk of loss of social esteem, status, or acceptance (Dickerson, Gruenewald, & Kemeny, 2004; Dickerson & Kemeny, 2004). Everyday events of social exclusion can include overt rejection or devaluation by individuals in a desired relationship, such as a friend, romantic partner, or a group.

Signals of potential social exclusion activate both dACC and AI (for reviews, see Eisenberger, 2012; Rotge et al., 2014). For example, in a negative social evaluation paradigm a confederate provided participants with rejecting, neutral, or accepting feedback (Eisenberger, Inagaki, Muscatell, Byrne Haltom, & Leary, 2011). Lower state levels of self-esteem were related to higher dACC and AI activation. In other work, rejection-themed images, which are signals of potential exclusion, prompted more dACC and AI activation than did acceptance-themed images (Kross, Egner, Ochsner, Hirsch, & Downey, 2007). Similarly, bereaved participants showed more dACC and AI activation to pictures of deceased relatives than to pictures of strangers, putatively as a result of eliciting grief related to loss (Gündel, O'Connor, Littrell, Fort, & Lane, 2003; O'Connor et al., 2008). When viewing videos of facial expressions, rejection sensitivity was positively correlated with dACC activation for disapproving expressions, an instance representing possible devaluation of the participant, but was unrelated to dACC activation for angry or disgusted expressions (Burklund, Eisenberger, & Lieberman, 2007). In response to pictures of smiling baby faces, women who lost an unborn child showed greater dACC activation than did participants who delivered a healthy child (Kersting et al., 2009). Taken together, these findings are highly indicative of a relationship between social pain associated with social exclusion or loss and dACC and AI activation, two brain areas previously related to the affective component of physical pain (Eisenberger, 2012, 2015).

Social Exclusion during the Cyberball Paradigm

The most widely used paradigm for investigating social exclusion is the Cyberball task (Williams, Cheung, & Choi, 2000). During the Cyberball task, a participant is commonly led to believe that he or she is playing a ball-tossing game on the computer with two other participants. In reality, the participant is not actually playing with other players but is instead interacting with two computer-controlled actors ("cyber-stooges"). The participant experiences two separate phases: an inclusion phase and an exclusion phase. During the inclusion phase, the participant and two cyber-stooges pass the ball between each other, and the participant receives the ball approximately one third of the time. In the exclusion phase, the ball is passed between the cyber-stooges, and the participant is excluded from the game. Comparison of the effects of the exclusion phase to the inclusion phase is then used to make inferences regarding social exclusion.

The first fMRI study using the Cyberball paradigm reported greater dACC and AI activation during the exclusion phase than during the inclusion phase, and dACC activation correlated positively with a self-report measure of social distress that assessed feelings of rejection, devaluation, and being disliked (Eisenberger, Lieberman, & Williams, 2003). Higher social distress following exclusion is consistent with exclusion as a type of SET, as self-reported devaluation and rejection increased following social exclusion. Social exclusion elicited dACC and AI activation, which are also observed as a manifestation of social pain (Eisenberger, 2012, 2015). Right ventral prefrontal cortex activation (RVPFC) was also observed during exclusion and correlated negatively with a self-report measure of social distress and dACC activation, suggesting a self-regulatory role in disrupting dACC activation. That is, RVPFC may mitigate dACC activation in response to social exclusion. Taken together, these findings indicate that social exclusion is associated with activation in brain regions similar to those associated with social and physical pain.

A recent meta-analysis of 46 studies (940 healthy participants) that investigated social pain as a result of social threat, exclusion, rejection, loss, or negative evaluation examined the extent to which regions of ACC activation were correlated with social pain (Rotge et al., 2014). Social pain was related to activation of the subgenual ACC, pregenual ACC, and anterior midcingulate cortex (part of dACC), and greater activation in these three regions was associated with greater self-reported distress. When examining only those studies using the Cyberball task (19 studies that included 467 healthy participants), all three regions – subgenual ACC, pregenual ACC, and anterior midcingulate cortex – showed activation in response to social exclusion. Greater activation of dACC following social exclusion during the Cyberball task has also been related to lower self-esteem (Onoda et al., 2010), an anxious attachment style (DeWall et al., 2012), lower neighborhood socioeconomic status (Gonzalez, Beckes, Chango, Allen, & Coan, 2015), high trait interpersonal hypersensitivity (Eisenberger, Way, Taylor, Welch, & Lieberman, 2007), and a tendency to feel socially disconnected on a daily basis (Eisenberger, Gable, & Lieberman, 2007). Reduced dACC activation is associated with early life stress (Puetz et al., 2014), presence of social support (Masten, Telzer, Fuligni, Lieberman, & Eisenberger, 2012), and an avoidant attachment style (DeWall et al., 2012). Taken together, these findings provide support for the involvement of dACC in social exclusion, the sensitivity of dACC activation to constructs putatively associated with vulnerability to social pain, and the tenability of the Cyberball paradigm as an effective task for examining social exclusion.

A modified version of the Cyberball task has been used to examine social exclusion in patients with schizophrenia (Gradin et al., 2012). Healthy controls were found to show increased activation of medial frontal gyrus (MFG) that was associated with increases in the degree of exclusion. However, neither MFG activation nor MFG activation computed with a difference score between exclusion and inclusion were found to correlate with a self-report measure of distress. Although patients with schizophrenia did not exhibit increased activation of MFG in response to exclusion, decreases in the MFG difference score correlated with increases in measures of positive symptoms. The typical ACC and AI activation observed in Cyberball studies was not observed in patients or controls, possibly as a result of the parametric fluctuation of exclusion over the course of 17 separate blocks (see Gradin et al., 2012). Given the observed relationship between MFG activation associated with social exclusion and symptom severity, these findings provide some support for the notion that stress elicited by SET may relate to symptom severity in schizophrenia. However, in light of the unique task parameters of the study, limiting comparison to previous work on the relationship between dACC and social exclusion, the proposed study will use a more conventional Cyberball design to probe dACC responses to social exclusion in schizophrenia.

Considering the role of SET as a stressor, dACC and AI activation elicited during social exclusion in the Cyberball task may relate to increases in pro-inflammatory cytokines, which represent a biological mechanism for how stress influences health (Baker, Nievergelt, & O'Connor, 2012; Miller, Chen, & Cole, 2009; Rohleder, 2014). In one study, participants completed the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) and separately completed the Cyberball task (Slavich, Way, Eisenberger, & Taylor, 2010). Increases in inflammatory activity elicited by the TSST were positively correlated with dACC and AI activation during social exclusion in the Cyberball task, providing support for the role of dACC and AI in stress elicited by social exclusion. Results obtained from Cyberball studies examining cortisol responses following social exclusion have been inconsistent (Geniole, Carré, & McCormick, 2011; Seidel et al., 2013; Zöller, Maroof, Weik, & Deinzer, 2010; Zwolinski, 2012). Cortisol responses appear to be only modestly elevated following social exclusion in women, particularly during the luteal phase (Zwolinski, 2012). Given the brief duration of social exclusion in the Cyberball, however, the task may not be sufficient in duration or intensity to elicit a consistent and robust cortisol response (Seidel et al., 2013; Zwolinski, 2012). Despite that dACC and AI activation following social exclusion appear related to potentiated inflammatory response, inconsistent cortisol findings following exclusion suggest that social exclusion during the Cyberball may not be a potent stressor.

dACC-Mediated Exclusion-Related Activation

During the Cyberball task, dACC activation following exclusion may be attributed to an expectancy violation, social distress, or a combination of both (Eisenberger, 2012, 2015; Eisenberger & Cole, 2012; Eisenberger & Lieberman, 2004; Spunt, Lieberman, Cohen, & Eisenberger, 2012). When there is an expectation of inclusion, an expectancy violation occurs when the participant is excluded given the discrepancy between the expectation and the actual outcome. Additionally, dACC activation during exclusion while performing the Cyberball task has been reliably associated with self-reports of social distress (i.e., feeling rejected, meaningless, or disliked; e.g., Eisenberger et al., 2003; Masten et al., 2009).

Interpreting dACC as a discrepancy detector is consistent with research on the conflict monitoring model of cognitive control. According to this computational model, the detection of conflict by dACC serves to recruit cognitive resources to improve or guide subsequent behavior

(Botvinick, Carter, Braver, Barch, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004; Carter & van Veen, 2007). dACC is activated by conflict and prompts dorsolateral prefrontal cortex (DLPFC; Botvinick et al., 2001; Durston et al., 2003; Egner & Hirsch, 2005a, 2005b; Kerns et al., 2004) and right ventrolateral prefrontal cortex to improve performance (Egner, 2011). DLPFC minimizes conflict by biasing posterior systems, such as parietal cortex (Corbetta & Shulman, 2002; Desimone & Duncan, 1995), which enhances attentional focus and cognitive control (Banich, 2009; Egner & Hirsch, 2005a; Silton et al., 2010). Enhanced attention then serves to reduce further conflict activation by biasing attention away from irrelevant information. Taken together, the conflict monitoring model implicates dACC, as a conflict monitor, and DLPFC, as the cognitive control implementer, in the adaptation of control following conflict.

Social exclusion during the Cyberball task may also prompt increases in social distress, by potentially reducing feelings of belongingness and self-esteem (Williams et al., 2000). As an SET, social exclusion is an indication of possible devaluation by group members and thus of risk of loss of social self-esteem, status, or acceptance (Dickerson et al., 2004; Dickerson & Kemeny, 2004). Increases in social distress as a result of social exclusion have been associated with a cascade of responses including increases in stress, negative self-evaluation, and such negative affect as fear, sadness, and shame (Dickerson et al., 2004; Kemeny, Gruenewald, & Dickerson, 2004). In turn, social distress may lead to adaptive behaviors to aid in reintegration to the group and increase acceptance from others (Denson, Spanovic, & Miller, 2009; Gonzaga, Keltner, Londahl, & Smith, 2001; Williams & Sommer, 1997) or to social withdrawal and isolation (Dickerson et al., 2004; Kemeny et al., 2004).

Support for an association between social distress and dACC activation relies on the affective neuroscience literature examining the role of dACC in pain affect (Apkarian, Bushnell, Treede,

& Zubieta, 2005; Eisenberger & Lieberman, 2004; Shackman et al., 2011). Meta-analytic studies indicate that measures of negative affect (Mechias, Etkin, & Kalisch, 2010) and pain (Farrell, Laird, & Egan, 2005; Vogt, 2005) are related to dACC activity. Similar to the conflict monitoring model, which suggests that dACC serves to elicit control, dACC may also engage adaptive control in response to negative affect and pain (Etkin, Egner, & Kalisch, 2011; Shackman et al., 2011). Indeed, cognitive and affective information interact and appear to recruit adaptive control in a similar fashion by prompting activation of DLPFC and inferior frontal gyrus (Pessoa, 2009).

Some evidence supporting the role of dACC in adaptive control in response to affectively salient information comes from studies observing monkeys with a lesioned cingulate sulcus, the monkey analogue to the rostral cingulate zone in dACC (Rudebeck, Buckley, Walton, & Rushworth, 2006). Monkeys with a lesioned cingulate sulcus were observed to be less likely to pick up food in the presence of fear-inducing stimuli than were controls, suggesting a mediating role of dACC between threat and adaptive behavior. Likewise, as threat becomes more imminent there is increased activation of monkey cingulate sulcus and an engagement of adaptive behaviors (Kalin, Shelton, Fox, Oakes, & Davidson, 2005). In humans, dACC appears similarly involved in adaptive defensive behaviors in response to potential or imminent danger (Mobbs et al., 2009; Mobbs et al., 2007) and in the correct encoding of the misperception of threat during the reversal of learned fear (Schiller, Levy, Niv, LeDoux, & Phelps, 2008), supporting the involvement of dACC in adaptive control during instances of threat (Shackman et al., 2011).

dACC sensitivity to both cognitive and affective information is further supported by studies investigating the association between conflict monitoring and negative affect. For example, a recent study examined the extent to which dACC activation during performance of a stop signal task was related to self-reported negative affect (Spunt et al., 2012). Error-related dACC activation was associated with within-subject variability of self-reported levels of frustration, but not with ratings of anxiety or unpleasantness.

These fMRI findings are consistent with event-related potential (ERP) studies supporting a relationship between ACC-mediated, conflict-related, ERP components—the error-related negativity (ERN) and conflict N2 (for review, see Larson, Clayson, & Clawson, 2014)—and state negative affect (Hajcak, McDonald, & Simons, 2004; Luu, Collins, & Tucker, 2000; Wiswede, Münte, & Rüsseler, 2009), trait anxiety (Larson, Clawson, Clayson, & Baldwin, 2013; Olvet & Hajcak, 2009; Weinberg, Klein, & Hajcak, 2012), fear (Danielmeier, Wessel, Steinhauser, & Ullsperger, 2009), sadness (Olvet & Hajcak, 2012), and depressive symptoms (Clawson, Clayson, & Larson, 2013; Larson et al., 2013). In addition, ACC has been implicated in the generation of both ERN (Brazdil, Roman, Daniel, & Rektor, 2005; Bush, Luu, & Posner, 2000; Stemmer, Segalowitz, Witzke, & Schonle, 2004; van Veen & Carter, 2002) and conflict N2 (Ladouceur, Dahl, & Carter, 2007; van Veen & Carter, 2002; Yeung, Botvinick, & Cohen, 2004), suggesting that ERN and conflict N2 represent manifestations of ACC activation. When participants completed a flanker paradigm following the Cyberball task, participants who were excluded recently displayed smaller ERN than those participants who just completed the inclusion block, suggesting that adaptive control following social distress may downregulate putative ACC activation (Themanson, Ball, Khatcherian, & Rosen, 2014). Considering the relationship between manifestations of ACC activation associated with conflict monitoring and affect-related measures, dACC response to expectancy violations and pain affect may serve a similar function during the Cyberball task, which would be to prompt adaptive control (Botvinick, 2007; Shackman et al., 2011; Weinberg et al., 2012).

Aside from involvement of dACC in discrepancy detection or changes with social distress, dACC is implicated in processes such as conflict monitoring (e.g., Botvinick, 2007; Botvinick et al., 2001), reinforcement learning (e.g., Holroyd & Coles, 2002, 2008; Holroyd, Yeung, Coles, & Cohen, 2005), response selection and evaluation (e.g., Banich, 2009; Silton et al., 2010), and prediction of response outcomes (e.g., Brown, 2013). All of these mechanistic accounts of dACC function to some degree implicate dACC in attentional control. That is, these accounts propose that dACC prompts top-down control processes to adjust behavior in a goal-directed fashion. A goal of the proposed study is to identify whether dACC activation following social exclusion is primarily the result of increased negative affect induced by social exclusion, the result of discrepancy detection, or a combination of the two. Considering dACC sensitivity to both discrepancy detection and increases with social distress, conflict-related ERPs may provide dependable measures of exclusion-related, dACC activation that are sensitive to expectancy violations and negative affect.

ERP Evidence for Discrepancy Detection and Social Distress

In order to identify dACC activation associated with discrepancy detection and social distress, the neural time course of dACC activation will be analyzed in the proposed study using ERPs, which offer a temporal resolution on the order of milliseconds. Identifying the temporal course of dACC activation has great potential to advance understanding of the role of dACC in social exclusion by identifying when dACC activation relates to discrepancy detection or to social distress or when discrepancy detection and social distress operate in conjunction.

Discrepancy Detection. An anterior N2 component elicited by visual stimuli is consistently related to novelty, conflict monitoring, and cognitive control (Folstein & Van Petten, 2008) and is proposed to be a manifestation of ACC-mediated conflict activation during exclusion events in the Cyberball task (Themanson, Khatcherian, Ball, & Rosen, 2013; Themanson et al., 2015). N2 is a scalp-negative deflection in the ERP with a fronto-central scalp distribution that peaks approximately 250-350 ms following stimulus presentation (Folstein & Van Petten, 2008; Larson et al., 2014; Yeung et al., 2004). During the Cyberball task, early exclusion-related activity in the ERP time window commonly associated with N2 has been examined and shown to be sensitive to social exclusion. Specifically, larger (more negative) N2 was observed during exclusionary ball throws than to inclusionary ball throws (Gutz, Küpper, Renneberg, & Niedeggen, 2011; Themanson et al., 2013; Themanson et al., 2015). Increased N2 activity following exclusionary throws, therefore, may index a discrepancy between the expectations of inclusion and the actual outcome of exclusion. Considering that source-localization studies have not yet examined whether ACC is the neural generator of N2 observed in the Cyberball, the proposed study will also investigate the extent to which ACC is implicated in N2 generation following exclusion events.

In light of findings of a relationship between increased ACC activation and increased self-reported social distress during the Cyberball task (Eisenberger et al., 2003), it is unclear why N2 elicited during exclusion phases does not appear to be related to self-reported distress (Themanson et al., 2013; Themanson et al., 2015). One possibility is that N2 is related to discrepancy detection following exclusion events rather than the social distress following exclusion (see Themanson et al., 2013; Themanson et al., 2015). This interpretation is supported by findings of similar N2 amplitude during both exclusionary throws in the exclusion phase and exclusionary throws during the inclusion phase. Thus, the proposed study will test whether N2 represents discrepancy detection and the extent to which N2 is associated with social distress.

Social distress. In Cyberball studies using motion for ball throws, sustained positive ERP activity, referred to subsequently as the exclusion slow potential (exclusion SP), has been observed with onset latency between 420 and 500 and lasting until 900 ms over fronto-central leads (Crowley et al., 2009; Crowley, Wu, Molfese, & Mayes, 2010; McPartland et al., 2011). Exclusion SP amplitude is more negative to exclusionary throws during the exclusion phase than to exclusionary throws during the inclusion phase and has been interpreted to reflect facilitated attention to exclusion events. Exclusion SP amplitude during exclusionary throws in both the inclusion phase and the exclusion phase were positively correlated with self-report measures of social distress (Crowley et al., 2009; Crowley et al., 2010). An ERP source-localization study in children implicated cortical regions, such as the subgenual cingulate cortex, ventral posterior cortex, insula, ACC, and posterior cingulate cortex in exclusion SP generation following social exclusion during the Cyberball (Crowley et al., 2010). Thus, exclusion SP seems to represent, in part, a manifestation of ACC activation and appears to be sensitive to increases with social distress.

Another ERP component related to measures of social distress following social exclusion is P3. P3 is the most widely investigated ERP component in studies involving Cyberball. P3a has a fronto-central scalp distribution, peaks between 250 and 500+ ms, and is associated with stimulus-driven orientation of attention processing, with larger amplitude indicating increased attention (Polich, 2007). P3a amplitude is observed to be larger for exclusionary throws during the exclusion phase than for exclusionary throws during the inclusion phase in children (Crowley et al., 2010). Additionally, P3a was observed as larger to exclusionary throws during an inclusion phase than during an exclusion phase, possibly due to the novelty of exclusion during the inclusion phase (Themanson et al., 2015). P3a is also observed to habituate to repeated stimulus presentations (Polich, 2007; Simons, Graham, Miles, & Chen, 2001).

The other subcomponent of P3, P3b, has a parietal scalp distribution and somewhat longer latency than P3a. P3b is sensitive to attentional engagement to a stimulus and represents processes such as context-updating and subsequent memory storage (Donchin, 1981; Polich, 2007). In one Cyberball study, P3b amplitude was larger during exclusionary throws in the exclusion phase than during the inclusion phase, suggesting greater allocation of attention to exclusionary throws in the context of social exclusion (Themanson et al., 2013). Considering that P3b is related to the updating of mental schemas (Donchin, 1981), another possible explanation for P3b activation following exclusion events is discrepancy between actual exclusion and expected inclusion based on the mental schema developed during social inclusion. The possibility of larger P3b to exclusionary throws during social exclusion than to exclusionary throws during inclusion has been subsequently explored in a single study and did not reveal any consistent associations (Themanson et al., 2015).

Summary of ERP findings. Based on the extant research literature, ERP findings obtained during the Cyberball task are encouraging and suggest important directions for future research. The ERP component that most consistently exhibits differential activity during inclusion and exclusion phases is exclusion SP (Crowley et al., 2009; Crowley et al., 2010; McPartland et al., 2011). This phenomenon likely relates, in part, to increases with social distress following exclusionary events. Although anterior N2 showed consistent activation to exclusionary throws, similar amplitudes were observed between exclusion and inclusion phases. Thus, it appears that anterior N2 amplitude may simply represents a manifestation of the overall discrepancy monitoring processes with increased activity following discrepancy detection. If anterior N2 were sensitive to social exclusion, then it would be expected that N2 would also increase during exclusion relative to inclusion phases. Source-localization findings of anterior N2 implicate ACC as the neural generator in conflict-inducing paradigms (Folstein & Van Petten, 2008; Ladouceur et al., 2007; van Veen & Carter, 2002; Yeung et al., 2004). However, the extent to which ACC contributes to anterior N2 activation during the Cyberball task remains unclear.

Uncertainties and inconsistencies associated with the ERP findings described above may be the result of methodological limitations, as ERP studies have required significant modifications to the Cyberball task that had been optimized for fMRI. In future examinations, it could be expected that N2 activation will be observed to exclusionary throws. It is also expected that exclusion SP will be increased following exclusionary throws during social exclusion than following exclusionary throws during inclusion and will relate to levels of self-reported social distress. It is likely that N2 is a manifestation of early exclusion-related ACC activation and that exclusion SP and P3 represent manifestations of late exclusion-related ACC activation. Given the functional significance of these ERP components, early exclusion-related ACC activation may be related to discrepancy detection that is not sensitive to changes with social distress, whereas late exclusion-related ACC activation seems related to changes with social distress.

Study Aims and Hypotheses

The present investigation is designed to develop a Cyberball task that is optimized for the recording of ERPs in healthy individuals and provides a future opportunity to examine SET in patients with schizophrenia. Stress elicited by social exclusion, a type of SET, may help to reveal a mechanism by which social environments influence symptoms and relapse, while greater sensitivity to social exclusion may be associated with a stronger relationship to symptom exacerbation.

Aim 1

The first aim is to identify the functional significance of early exclusion-related ERP activity in the Cyberball task.

Hypothesis 1a. It is hypothesized that an N2 will be observed that will be larger following exclusionary throws than following inclusionary throws. N2 amplitude should be similar following exclusionary throws during inclusion and exclusion phases. This hypothesis is based on findings indicating a similar pattern of activity (Themanson et al., 2013; Themanson et al., 2015) and other findings implicating N2 in conflict monitoring processes (Folstein & Van Petten, 2008; Larson et al., 2014; Yeung et al., 2004).

Hypothesis 1b. Based on previous N2 findings (Themanson et al., 2013; Themanson et al., 2015), N2 activity is not expected to relate to any self-report measures of social distress.

Hypothesis 1c. Source localization of early exclusion-related ERP activity is expected to implicate a neural generator located in ACC. This finding would support the role of N2 in discrepancy detection, as ACC is implicated in conflict detection (Larson et al., 2014; Yeung et al., 2004).

Aim 2

Next, the functional significance of late exclusion-related ERP activity, which includes exclusion SP, frontal P3 (P3a), and parietal P3 (P3b), will be established.

Hypothesis 2a. As previously observed (Crowley et al., 2009; Crowley et al., 2010; McPartland et al., 2011), exclusion SP should be larger to exclusionary throws than to inclusionary throws. Furthermore, exclusion SP should be larger to exclusionary throws during the exclusion phase than to exclusionary throws during the inclusion phase.

Hypothesis 2b. Some studies have shown that frontal P3 (Crowley et al., 2010) and parietal P3 (Themanson et al., 2013) are larger for exclusionary throws during the exclusion phase than during the inclusion phase. A similar relationship is expected in the proposed study.

Hypothesis 2c. Exclusion SP and frontal P3 amplitudes to exclusionary throws during the exclusion phase should be positively correlated with self-report measures of social distress. Such findings would provide support for the notion that late exclusion-related ERP activity is associated with changes in social distress following exclusion.

Hypothesis 2d. Late exclusion-related EEG activity, which includes the temporal windows for exclusion SP, frontal P3, and parietal P3, elicited following exclusionary throws should have a neural generator in ACC. If late exclusion-related activity is sensitive to changes with social distress following social exclusion, then it is expected to have at least one neural generator in ACC. This hypothesis is supported by a previous ERP source localization study in children that implicated ACC as one of the neural generators of late exclusion-related activity (Crowley et al., 2010).

Hypothesis 2e. High levels of social support should be related to diminished exclusion SP and frontal P3. This hypothesis is consistent with fMRI work showing reduced ACC activation to social exclusion in individuals with high levels of social support (Masten et al., 2012).

Method

Participants

Initial study enrollment included 41 participants. Participants were recruited from introductory psychology undergraduate courses and received course credit for their participation. Exclusion criteria included alcohol or substance dependence/abuse, any previous or present diagnosis of a psychiatric disorder, neurological disorder, head trauma, learning disability, or limited fluency in English. Written informed consent was obtained from all participants.

Following study enrollment, four participants were excluded for familiarity with the Cyberball paradigm and its purpose, three participants were excluded due to hardware malfunction, one participant was excluded for a possible psychiatric diagnosis, and one participant was excluded for having an insufficient number of trials retained for averaging following artifact correction and rejection (see Electrophysiological Data Recording and Reduction section). Thus, the final study sample included 32 participants (22 females, 10 males).

The average age of participants was 20 years with a standard deviation of 2 years (range: 18 to 26 years). The ethnic distribution of the final sample included 2 African Americans, 13 Asian Americans, 6 Caucasians, 8 Hispanics, 1 Pacific Islander, and 2 multi-ethnic participants. For all analyses that examined the first block of the exclusion phase, 3 of 32 participants were removed. These two female participants and one male participant were excluded due to having too few trials to meet the reliability cutoffs shown in Table 1. Thus, this subsample contained data for 29 participants. For the source waveform analyses in the Appendix, one male participant was excluded for having some source waveform scores greater than 5 *SD*s away from the mean. Source waveform analyses were conducted on the remaining 31 participants.

Sensitivity power analyses were conducted using G*Power (v3.1.9.2) and estimated the minimal detectable effect sizes (Faul, Erdfelder, Buchner, & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007). Minimal detectable effect sizes were computed using a power level of .80, an alpha level of .05, and a sample size of 32. For differentiating between two dependent means, the minimal detectable effect size was a Cohen's *d* of .51. For assessing the relationships between two variables, the minimal detectable effect size was a Pearson's *r* of .46.

Measures

Negative and positive affect. To measure trait levels of negative and positive affect, the Negative Temperament Scale (GTS-NTS) and Positive Temperament Scale (GTS-PTS) of the General Temperament Survey were administered (Watson & Clark, 1993). The GTS-NTS and GTS-PTS consist of true or false statements regarding the participant's attitudes, feelings, and interests. The GTS-NTS contains items such as "My anger frequently gets the best of me" and "Little things upset me too much", and the GTS-PTS contains items such as "I live a very full life" and "I am usually alert and attentive".

To assess state levels of negative and positive affect, the Positive and Negative Affect Schedule (PANAS) was administered (Crawford & Henry, 2004; Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item measure wherein participants respond regarding how they currently feel. The negative affect scale contains items such as "upset" and "rejected", whereas the positive affect scale contains items such as "enthusiastic" and "alert". Participants rate items on a 5-point Likert scale from 1 (*not at all*) to 5 (*extremely*).

Additionally, the Need-Threat Scale (NTS) was administered to assess social distress (Masten et al., 2009; van Beest & Williams, 2006; Williams et al., 2000; Zadro, Williams, & Richardson, 2004). The NTS is 20-item measure that includes four subscales: belongingness ("I felt rejected"), self-esteem ("I felt liked"), control ("I felt powerful"), and meaningful existence ("I felt invisible") and is commonly used to assess the participant's affective experience during the Cyberball task. Research using the NTS has observed a relationship between distress and dACC activation during social exclusion in the Cyberball task (e.g., Eisenberger et al., 2003; Onoda et al., 2009; Onoda et al., 2010).

A brief mood check was also administered following the completion of the Cyberball task. Consistent with prior work using the Cyberball task (Eisenberger et al., 2003; Gonsalkorale & Williams, 2007; Williams et al., 2000; Zadro et al., 2004), participants rated items on a 7-point Likert scale from 1 (*Bad*) to 7 (*Good*), 1 (*Sad*) to 7 (*Happy*), 1 (*Unfriendly*) to 7 (*Friendly*), and 1 (*Relaxed*) to 7 (*Tense*). The Relaxed/Tense item was reverse scored.

Anxiety and depression. In order to evaluate potential contributions of anxiety or depression, measures of anxiety and depressive symptoms were also administered. The Penn State Worry Questionnaire (PSWQ) was used to assess anxious apprehension (Meyer, Miller, Metzger, & Borkovec, 1990). On the PSWQ, participants rate the extent to which they identify with various statements (e.g., "I worry all of the time") on a 5-point Likert scale ranging from 1 (*not at all typical of me*) to 5 (*very typical of me*). Additionally, the Anxious Arousal and Anhedonic Depression subscales of the Mood and Anxiety Symptom Questionnaire (MASQ) was administered (Watson, Clark, et al., 1995; Watson, Weber, et al., 1995). On the MASQ, participants rate how much they experienced an item over the previous week on a 5-point Likert scale ranging from 1 (*not at all*) to 5 (*extremely*). The MASQ Anxious Arousal subscale (MASQ-AA) contains items such as "Hands were shaky" and "Had pain in my chest", and the MASQ Anhedonic Depression subscale (MASQ-AD) contains items such as "Felt withdrawn from other people" and "Felt like nothing was very enjoyable".

Social support. To measure social support, the Social Support Questionnaire (SSQ) was administered to assess both perceived number of individuals from which the participant receives social support and the participant's satisfaction with the received social support (Sarason, Levine, Basham, & Sarason, 1983). The SSQ is a 6-item measure that asks participants to first identify whom they can rely on for support in a given situation (e.g., "Whom can you really count on to be dependable when you need help?"). Then participants rate the degree of satisfaction with the overall support they receive on a 6-point Likert scale ranging from 1 (*very dissatisfied*) to 6 (*very satisfied*).

Manipulation check. To assess the extent to which participants perceived themselves being excluded during the exclusion phase, participants indicated the percentage of throws that were directed to them during the last round of the ball-tossing game. They also rated two items ("I was ignored" and "I was excluded") on a 5-point Likert scale ranging from 1 (*Not at all*) to 5 (*Very much so*).

Experimental Task

Upon arriving at the lab, task procedures were explained and written informed consent was obtained. Participants were introduced to a female confederate, who was in the process of having an EEG cap applied. Participants subsequently were photographed and completed the self-report measures. Following completion of these questionnaires, EEG electrodes were applied, and a Brain Products digitizer was used to determine the 3-dimensional positions of each EEG electrode. Participants then completed the Cyberball task.

Cyberball task. The examiner explained to participants that they would be playing a virtual ball-tossing game with two other participants. However, in reality the participants played a computer program with predetermined events. Each task began with a display that imitated the game making connections with two other participants. Photos of each participant were displayed on the screen with the name of the player and confederates printed above the photos and the status of each player printed below the photos (e.g., "Completing practice task" or "Ready"). For the confederates, one photo was of the female confederate that the participant had just met, and the other photo was of a male confederate. The participants were instructed that the male confederate was in another testing room across the hall. While waiting for the other participants to seemingly indicate that they were ready, the task instructions were explained, and a practice version of the task was completed.

Following these instructions, a display with three baseball mitts placed in three corners of an imaginary equilateral triangle appeared. The baseball mitt representing the participant was placed in the bottom center of the screen. The two ‘cyber-stooges’ were placed at the two other corners. To practice passing the ball, the participant completed 20 practice trials. Participants used a left-index button press to pass the ball to the mitt on the left and a right-index button press to pass the ball to the mitt on the right. During a trial, the mitt that had the ball in its possession was indicated with the ball appearing next to that mitt. When a ball was passed, an arrow appeared indicating to which mitt the ball was thrown. After an arrow depicting the direction of the ball throw had been displayed for 1,000 ms, the ball then appeared next to the receiving mitt. During the practice phase the participant received the ball 33% of the time, and the cyber stooges threw the ball after a fixed 1,200 ms.

After completing the practice task to ensure understanding of the simulated play and after the other players seemingly finished completing their own practice tasks, the inclusion phase began. The inclusion phase consisted of 160 throws, and the participant received the ball 33% of the time. The exclusion phase then commenced and also consisted of 160 throws. To avoid the participants disengaging from the task, the participant pseudo-randomly received the ball once per every ten-trial set. During each trial of the inclusion and exclusion phases, the cyber stooges waited 200 to 1,500 ms (determined randomly) before passing the ball to mimic the time it would take an actual, human player to pass the ball.

Immediately after the exclusion phase, the participant completed the PANAS, NTS, brief mood items, and the manipulation check. Following completion of the questionnaires, the participant was debriefed, including being fully informed about the use of deception. In order to ensure that participation was educational, the purpose of the Cyberball task and how it relates to real-world phenomena was also discussed during debriefing.

Electrophysiological Data Recording and Reduction

The electroencephalogram (EEG) was recorded from 96 active Ag/AgCl electrodes on the scalp using an ActiCHamp system (Brain Products Inc.). EEG recordings were referenced to the left mastoid during data acquisition. Electrooculogram (EOG) was recorded by placing 6 of the 96 electrodes above and below the left and right eyes as well as near the outer canthi of the eyes. All impedances were maintained below 20 kΩ. Data were digitized continuously at 1,000 Hz and used a bandpass of 0.05 to 200 Hz. Data were rereferenced offline to averaged mastoids and digitally high- and low-pass filtered at .05 (6 dB/oct) and 8 Hz (24 dB/oct), respectively, using a zero-phase Butterworth filter.

Stimulus-locked epochs for inclusionary and exclusionary events were extracted from 200 ms prior to the presentation of the arrow indicating the direction of the ball pass to 1,000 ms following the presentation of the arrow. The first 200 ms of that epoch served as a prestimulus baseline. In order to remove ocular artifact, individual artifact templates corresponding to eye blinks and saccades were created. An adaptive artifact correction approach using the individual-subject artifact templates was then implemented (Ille, Berg, & Scherg, 2002) using Brain Electrical Source Analysis (BESA) software (Version 6.1, Megis Software, Gräfeling Munich). Following ocular artifact correction, trials that exceeded more than ±90 µV in any channel were rejected.

Following artifact correction and rejection, individual-subject ERPs were analyzed. Electrode sites for analysis were chosen based on scalp distribution of the ERP components of interest and previous Cyberball ERP research (Crowley et al., 2009; Crowley et al., 2010; McPartland et al., 2011; Themanson et al., 2013; Themanson et al., 2015). See Figure 1 for the sensor layout of the 96 EEG recording sites. The positioning and nomenclature of the recording sites follow the published recommendations for high-density EEG recording (Oostenveld & Praamstra, 2001). Briefly, each site location is designated with a letter, such as 'F' for frontal, 'C' for central, 'P' for parietal, and 'O' for occipital. A 'z' denotes that the electrode is placed on an imaginary midline that runs between the nasion and inion. Even numbers following the letters refer to positions over the right hemisphere, and odd numbers refer to positions over the left hemisphere. Higher numbers denote positions further away from the midline. 'FCz' denotes a position that is halfway between Fz and Cz, whereas 'CPz' denotes a position that is halfway between Cz and Pz. 'C1h' denotes a position between Cz and C1, and 'CCP1h' denotes a position that is between C1h and CP1h.

Early exclusion-related activity corresponding to the N2 time window was extracted as the average ERP amplitude from 275 to 325 ms following stimulus presentation averaged across four sites: vertex point (Cz), CCP1H, CCP2H, and CPz (see Figure 1 for sensor layout). Late exclusion-related activity corresponding to the time window for frontal P3, parietal P3, and exclusion SP was also analyzed. The extent to which frontal P3 reflects P3a activity and parietal P3 reflects P3b activity is explored in the Discussion section. Frontal and parietal P3 amplitudes were extracted using an adaptive mean approach to avoid the biasing effects of background EEG noise as well as to allow for individual-subject latency variability (Clayson, Baldwin, & Larson, 2013; Luck, 2014). Frontal P3 amplitude was extracted as the average ERP amplitude from 15 ms pre-peak to 15ms post-peak positive amplitude between 300 and 500 ms following stimulus presentation averaged across three sites: FFC1H, FFC2H, and FCz. Parietal P3 amplitude was measured as the average ERP amplitude from 15 ms pre-peak to 15ms post-peak positive amplitude between 250 and 450 ms following stimulus presentation averaged across five sites: Pz, P1, P2, PPO1H, and PPO2H. Exclusion SP amplitude was scored as the average amplitude between 600 and 900ms averaged across six sites: Fz, F1, F2, FCz, FC1, and FC2.

In order to ensure that ERP score averages were reliable, the number of trials needed to achieve a reliability threshold of .70 was calculated for each condition. A reliability threshold of .70 was deemed acceptable based on published guidelines for ERP score reliability for paradigms that are in the early stages of development (Clayson & Miller, 2017b). Since the present manuscript examines a newly modified Cyberball paradigm that was optimized for ERP research, this paradigm was considered in the early stages of development and used the .70 reliability threshold. ERP score reliability as a function of the number of trials and event type needed for a stable average for each ERP component was examined using the ERP Reliability Analysis (ERA) Toolbox v 0.3.4 (Clayson & Miller, 2017a). The ERA Toolbox calculated score dependability based on algorithms from generalizability theory and used CmdStan v 2.14.0 (Stan Development Team, 2017) to implement the analyses in Stan (Carpenter et al., 2017). Table 1 summarizes the number of trials needed to obtain a dependability point estimate of .70 for each ERP component and event type. As mentioned above, one participant had insufficient trials for all trial cutoffs and was excluded. Overall dependability estimates and their 95% credible intervals for ERP score averages used the mean number of trials retained for each event type after removing the participant with insufficient trials. Overall ERP score dependability estimates and their associated 95% credible intervals are presented in Table 1. The average ± standard deviation (range: minimum to maximum) number of trials for each event type were 50 ± 4 (range: 34 to 55) for inclusionary throws during the inclusion phase, 51 ± 4 (range: 36 to 56) for exclusionary throws during the inclusion phase, and 107 ± 19 (range: 38 to 128) for exclusionary throws during the exclusion phase.

BESA Source Localization

In order to model the neural time course of exclusion-related activation, source localization was conducted using BESA software. To model early exclusion-related activation, EEG data from the time window corresponding to the mean amplitude window for N2 amplitude extraction were used. To model late exclusion-related activation, EEG data for the time windows corresponding to the amplitude measurement windows for frontal and parietal P3 as well as the mean amplitude window for exclusion SP were used. A discrete multiple source model was created using regional sources to model scalp EEG activity. BESA regional sources comprise three equivalent current dipoles with identical spatial locations but mutually orthogonal orientations, thus modeling current flow in any direction from the specified region (Scherg & Berg, 1996; Scherg & Von Cramon, 1986). Regional sources effectively model the activity of gray matter in the immediate vicinity of the source, which can lead to more reliable solutions in individual-subject data as the regional source will be more stable in noisy data than single dipoles (Paul-Jordanov, Hoechstetter, Bornfleth, Waelkens, & Scherg, 2016).

Prior to beginning source analyses, regions of interest for the present examination were first identified from fMRI Cyberball research. Although ACC was the primary region of interest, other regional sources were included to develop an adequate source model. Other candidate brain regions that have been observed in response to social exclusion were also modeled. These regions included right ventrolateral prefrontal cortex (vlPFC) and AI (Eisenberger et al., 2003; Masten et al., 2009) as well as right posterior cingulate cortex (PCC; Onoda et al., 2009).

To construct adequate source models of exclusion-related activation, a regional source was placed in dACC (Eisenberger et al., 2003). Then constrained bilateral, symmetric regional sources corresponding to AI and single regional sources for right vlPFC and right PCC (Onoda et al., 2009) were modeled in the grand-average data. After examining the source model, an additional regional source was placed in right superior frontal gyrus (SFG) to improve the modeling of frontal P3 activity, based on studies implicating right SFG in frontal P3 activity (Crottaz-Herbette & Menon, 2006; Galashan, Fehr, & Herrmann, 2015; Kiehl, Laurens, Duty, Forster, & Liddle, 2001). Three noise sources were also included in the model to account for some brain activity outside the regions of interest, residual ocular artifact, and background EEG noise. Without the noise sources, the regional sources of interest would have attempted to model the noise activity, thus decreasing the signal-to-noise ratio for the brains regions of interest. One regional source was placed to model residual ocular activity, and bilateral regional sources were placed in occipital cortices. For a visualization of the spatial locations of the regional sources, see Figure 2. Montreal Neurological Institute coordinates for each regional source are shown in Table 2.

Prior to fitting the source model, ERP data were digitally high- and low-pass filtered in BESA at .05 (6 dB/oct) and 8 Hz (24 dB/oct), respectively, using a zero-phase Butterworth filter. After regional sources were examined in the grand-average data, source analyses were performed separately for each individual based on the brain regions identified in the grand averages. Spatial locations of the regional sources from the grand averages were fixed for individual-subject source analyses. Source analyses on the individual-subject data computed the orientations of one of the three axes of the regional sources that minimized the residual variance between the modeled scalp data and the actual EEG data. In order to calculate the source waveforms for the models, a four-shell ellipsoidal head model (Berg & Scherg, 1994a) and a regularization constant of 1% for the inverse operator were used. One of the three axes of each regional sources was oriented to explain the maximal amount of variance in scalp EEG during the time windows of interest: early and late exclusion-related activity (see Table 2). Regional source orientations were adjusted separately for each event type and participant, and the source strength along the axis that had the highest source strength was used for subsequent statistical analyses.

After fitting the individual source models and orienting the regional sources, the source waveforms were baseline-adjusted using the 200 ms of prestimulus activity. Scoring windows were then determined based on visual inspection of the source waveforms and the temporal windows used for the analysis of the ERPs in sensor space. For dACC, two windows were used for scoring. The average activity from 15 ms pre-peak to 15 ms post-peak negative amplitude between 250 and 350 ms was used and corresponded to the N2 time window. The average activity from 15 ms pre-peak to 15 ms post-peak positive amplitude between 300 and 500 ms was used and corresponded to a frontal P3 time window. For PCC and SFG, the average activity from 15 ms pre-peak to 15 ms post-peak positive amplitude between 200 and 500ms was used and corresponded to a parietal P3 time window. For left and right AI source waveforms, two time windows were used. The average activity from 15 ms pre-peak to 15 ms post-peak negative amplitude between 250 and 375 ms was used and corresponded to the N2 time window. The average activity from 600 to 900 ms was used and corresponded to the exclusion SP time window. For right vlPFC source waveform, the average activity from 600 to 900 ms was used and corresponded to the exclusion SP time window.

Data Analysis

In order to avoid the biasing effects of non-normality and (co)variance heterogeneity as well as to reduce Type I error (see Dien, 2017; Dien & Santuzzi, 2005), robust analyses of variance (ANOVAs) were implemented within the ERP PCA Toolkit (Dien, 2010; Keselman, Wilcox, & Lix, 2003). The number of iterations used for bootstrapping was 50,000, and the starting seed for the random number generator was set to 1,000 for all analyses to ensure replicability (see Dien, Franklin, & May, 2006; Dien, Michelson, & Franklin, 2010). Although robust statistics are more conservative than conventional ANOVAs, *p*-values are interpreted in the same manner (Dien, 2017).

To assess whether the Cyberball paradigm impacted state negative or positive affect scores, a 2-Time (pre, post) x 2-Affect (negative, positive) robust ANOVA was performed for PANAS state negative and positive affect scores. Separate 2-Throw (inclusionary throw during inclusion phase, exclusionary throw during inclusion phase) robust ANOVAs were conducted for scalp-activity component amplitudes and for amplitude measurements of source waveforms. Then separate 2-Phase (exclusionary throw during inclusion phase, exclusionary throw during exclusion phase) robust ANOVAs were conducted for each ERP component, both peak sensor-space amplitudes and peak source-space amplitudes. Zero-order correlations were used to compare sensor- and source-space ERP component amplitudes for exclusionary throws during the exclusion phase to self-report measures of social distress, changes in state negative and positive affect, and brief mood items. When left or right AI activity and early or late dACC and AI activity significantly correlated with self-report measures, correlation coefficients were compared using tests for the equality of two dependent correlations to examine for possible lateralization and temporal dissociation effects (Steiger, 1980a, 1980b).

Results

Manipulation Check

Summary data for the self-report measures related to the manipulation check are located in Table 3. A robust Time x Affect ANOVA on PANAS state negative and positive affect scores revealed a main effect of time, indicating that self-reported levels of negative and positive affect were lower after completing the Cyberball task than they were prior to beginning the task, $T_{\mathrm{WJt}}/c(1.0,31.0)=101.14$, $p<.0001$. Overall, positive affect scores were higher than negative affect scores, as indicated by a main effect of affect, $T_{\mathrm{WJt}}/c(1.0,31.0)=101.88$, $p<.0001$. The Time x Affect interaction was also significant and indicated an ordinal interaction with a greater decrease in positive affect scores than in negative affect scores, $T_{\mathrm{WJt}}/c(1.0,31.0)=53.06$, $p<.0001$. Positive affect scores were consistently higher than negative affect scores before and after the Cyberball task, $T_{\mathrm{WJt}}/c(1.0,31.0)=141.24$, $p<.0001$; $T_{\mathrm{WJt}}/c(1.0,31.0)=29.78$, $p<.0001$, respectively. Positive and negative affect scores both decreased from before to after the Cyberball task, $T_{\mathrm{WJt}}/c(1.0,31.0)=89.55$, $p<.0001$; $T_{\mathrm{WJt}}/c(1.0,31.0)=23.57$, $p<.0001$, respectively. Participation in the Cyberball paradigm resulted in a decrease of PANAS state negative and positive affect from pre-task measurement to post-task measurement with a larger decrease observed for state positive affect scores than for state negative affect scores.

For the purpose of comparing the present study to other Cyberball studies, ostracism distress was computed as the average of items on the Need-Threat Scale (see Table 3). The mean ostracism distress score was rated as moderate ($M = 3.2$, $SD = 0.5$), which was similar to other samples of young adults ($M = 3.3$, $SD = 0.8$; $M = 3.0$, $SD = 0.7$; $M = 3.0$, $SD = 1.1$, respectively) (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006; Masten, Telzer, & Eisenberger, 2011; Xu et al., 2016). Participants reported receiving the ball an average of 16% ($SD = 8\%$) of throws during the exclusion phase of the task, which was a significantly higher estimate than the 11% of throws that participants actually received, *t*(31) = 3.34, *p* = .002, but significantly lower than the 33% of throws that were received in the inclusion phase, *t*(31) = -11.47, *p* <.0001. Based on the average scores of 3.3 (*SD*s = 1.2) for the items "I was ignored" and "I was excluded", participants reported being moderately ignored and excluded. For the brief mood items Bad/Good, Sad/Happy, Unfriendly/Friendly, and Relaxed/Tense, participants generally responded fairly neutrally (see Table 3).

Overall, participants reported a decrease in state negative and positive affect from pre-task to post-task measurements and feeling somewhat distressed, moderately ignored, and moderately excluded following the completion of the Cyberball paradigm. Participants also estimated receiving few ball throws in the exclusion phase of the paradigm.

Early versus Late Throws

Compared to previous Cyberball paradigms, a high number of exclusionary throws was used in order to obtain an adequate signal-to-noise ratio for evaluating the BESA source model. The results below indicated that the scalp- and source-waveform activity differed depending on whether the data from the first block of the exclusion phase or the data from the second block of the exclusion phase were used. In light of the changes in scalp- and source-waveform activity from the first block to the second block of the exclusion phase, it is possible that these changes in activity were due to participants disengaging from the task over time. This possibility and its justification are explored in detail in the Social Exclusion Manipulation section of the Discussion section. Given this possibility, data from the first block of the exclusion phase when participants were likely more engaged are analyzed below and emphasized throughout the Discussion section. Analyses for the data including all exclusionary throws during the exclusion phase are detailed in the Appendix.

The present Cyberball paradigm consisted of 160 exclusionary throws, and the numbers of throws used in other ERP Cyberball studies were 80 (Themanson et al., 2015), 50 (Themanson et al., 2013), 30 (Crowley et al., 2009), 36 (Crowley et al., 2010), and 44 (McPartland et al., 2011). P3b amplitude has been observed to decrease to exclusionary throws during the exclusion phase after the first 20 exclusionary throws of the exclusion phase (Themanson et al., 2015). To evaluate the impact of the high number of trials in the present data set, scalp- and source-space scores during the first 20 exclusionary throws were compared to the remaining exclusionary throws during the exclusion phase. Summary data for the scalp- and source-level activity scores are shown in Table 4, and grand-average waveforms are shown in Figures 3 through 5. The average ± standard deviation (range: minimum to maximum) number of trials were 18 ± 2 (range: 13 to 20) for exclusionary throws during the first block of the exclusion phase and 93 ± 9 (range: 67 to 108) for exclusionary throws during the second block of the exclusion phase.

First block versus second block comparisons. For scalp-space amplitudes, a robust ANOVA comparing N2 amplitudes during the first block of exclusionary throws during the exclusion phase to the second block of exclusionary throws during the exclusion phase indicated that N2 amplitudes were similar across blocks, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=2.62$, $p=.12$. This finding suggests that the length of the exclusion phase of this modified paradigm did not impact N2 amplitude scores.

Frontal P3 amplitudes were larger during the first block of the exclusion phase than during the second block of the exclusion phase, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=12.67$, $p=.001$. The robust ANOVA for parietal P3 amplitudes approached significance and indicated that parietal P3 amplitudes were somewhat larger during the first block of the exclusion phase than during the second block of the exclusion phase, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=5.62$, $p=.05$. Exclusion SP amplitudes were more negative for exclusionary throws during the second block of the exclusion phase than for exclusionary throws during the first block of the exclusion phase, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=9.19$, $p=.005$. Whereas frontal P3 (and to some extent parietal P3) was larger, exclusion SP was smaller during the first block than during the second block. These analyses suggest that the high number of trials included in the exclusion phase impacted the comparisons of scalp-space scores for the data averaging across all exclusionary throws during the exclusion phase. One explanation for the change in scalp-space scores may be a decrease in task engagement over the numerous exclusionary throws.

For source-space amplitudes, early dACC activity, which corresponds to the N2 time window, was greater during the first block of the exclusion phase than during the second block of the exclusion phase and was not consistent with N2 activity, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=6.38$, $p=.02$. Unlike frontal P3 activity, late dACC activity was similar across the blocks of the exclusion phase, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=2.81$, $p=.11$.

Consistent with N2 activity, early left and right AI activity was similar for exclusionary throws during the first block and for exclusionary throws during the second block, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=1.64$, $p=.21$; $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=0.10$, $p=.76$, respectively. Paralleling the exclusion SP findings and further supporting the role of these brain regions in exclusion SP generation, left and right AI activity as well as right vlPFC activity were greater for exclusionary throws during the first block than for exclusionary throws during the second block, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=20.44$, $p<.001$; $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=10.56$, $p=.003$; $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=4.76$, $p=.04$, respectively. PCC and SFG activity was greater during the first block than during the second block, $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=13.51$, $p=.002$; $T_{\mathrm{WJt}}/\mathrm{c}(1.0,28.0)=21.98$, $p<.001$, respectively. The pattern of PCC and SFG findings was similar to the pattern of parietal P3 findings, providing support for the role of PCC and SFG in parietal P3 generation. Similar to scalp-space amplitudes, the pattern of effects of source-space amplitudes suggests that the high number of trials included in the exclusion phase accounted for some of the decreased activity observed between exclusionary throws during the inclusion phase and all exclusionary throws during the exclusion phase (see Appendix for analysis of data including all exclusionary throws during the exclusion phase).

ERP Component Analyses

Grand-average scalp activity waveforms and topographic maps are presented in Figures 6 and 7. Summary data for scalp-activity component amplitudes are presented in Table 4.

N2. Contrary to the predicted pattern in hypothesis 1a, a robust ANOVA on N2 amplitudes for inclusionary and exclusionary throws during the inclusion phase indicated that N2 amplitude was more negative for inclusionary throws than for exclusionary throws, $T_{\mathrm{WJt}}/c(1.0,28.0)=8.64$, $p=.007$. Consistent with hypothesis 1a, N2 amplitudes for exclusionary throws were similar across the inclusion phase and the first block of the exclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=3.53$, $p=.07$. Such a pattern is suggestive of N2 not being sensitive to social exclusion.

Frontal P3. A significant difference between inclusionary and exclusionary throws during the inclusion phase was not observed for frontal P3 amplitude, as indicated by a nonsignificant robust ANOVA, $T_{\mathrm{WJt}}/c(1.0,28.0)=1.42$, $p=.24$. This finding is consistent with the role of frontal P3 in novelty and habituation, as inclusionary and exclusionary throws were equiprobable during the inclusion phase. Although it was expected that frontal P3 would be larger for exclusionary throws during the exclusion phase (hypothesis 2b), frontal P3 amplitude was similar for exclusionary throws during the inclusion phase and for exclusionary throws during the first block of the exclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=1.18$, $p=.29$.

Parietal P3. The robust ANOVA on parietal P3 amplitudes during the inclusion phase yielded a nonsignificant effect, indicating that similar amplitudes were observed for inclusionary and exclusionary throws, $T_{WJt}/c(1.0,28.0)<0.01$, p=.97. Similar activity across inclusionary and exclusionary throws for parietal P3 suggests that processes related to context-updating were similar for each throw type during the inclusion phase. Contrary to hypothesis 2b, parietal P3 amplitude for exclusionary throws during the inclusion phase were similar to parietal P3 amplitude for exclusionary throws during the first block of the exclusion phase, $T_{WJt}/c(1.0,28.0)=0.59$, p=.46.

Exclusion SP. A robust ANOVA on exclusion SP amplitudes for inclusionary and exclusionary throws during the inclusion phase indicated that exclusion SP amplitude was more negative for inclusionary throws than for exclusionary throws, $T_{WJt}/c(1.0,28.0)=23.64$, p<.001. Contrary to hypothesis 2a, exclusion SP amplitudes were similar for exclusionary throws during the inclusion phase and for exclusionary throws during the first block of the exclusion phase, $T_{WJt}/c(1.0,28.0)=0.98$, p=.33. This pattern of effects indicates that exclusion SP amplitude was not sensitive to the social exclusion manipulation.

Source Waveform Analyses

Grand-average source waveforms are presented in Figure 8. Summary data for source component amplitudes are presented in Table 4.

dACC. The first scoring window for dACC corresponded to scalp-recorded N2 activity. A robust ANOVA comparing source-waveform peaks during inclusionary and exclusionary throws during the inclusion phase indicated that dACC activity was larger for exclusionary throws than for inclusionary throws, $T_{WJt}/c(1.0,28.0)=7.58$, p=.01. For exclusionary throws, dACC activity during the inclusion phase was similar to activity during the first block of the exclusion phase,

$T_{WJt}/c(1.0,28.0)=0.71$, $p=.40$. Similar to the N2 pattern of findings, dACC differentiated between inclusionary and exclusionary throws in the inclusion phase and between exclusionary throws during the inclusion and exclusion phases. Given that dACC findings were similar to N2 findings, scalp N2 activity may represent a manifestation of dACC activity as predicted in hypothesis 1c.

The second scoring window for dACC corresponded to frontal P3 activity. Contrary to the pattern of findings observed for throws during the inclusion block for frontal P3, robust ANOVA indicated that activity to exclusionary throws during the inclusion phase was larger than activity to inclusionary throws during the inclusion phase, $T_{WJt}/c(1.0,28.0)=5.10$, $p=.04$. Paralleling the scalp frontal P3 findings, dACC activity for exclusionary throws during the inclusion phase was similar to dACC activity for exclusionary throws during the first block of the exclusion phase, $T_{WJt}/c(1.0,38.0)=0.34$, $p=.58$. However, the pattern of effects predicted in hypothesis 2d was that activity would be greater during the exclusion phase than during the inclusion phase.

Left AI. The first scoring window for the left AI source corresponded to N2 activity. Inconsistent with scalp N2 findings, a robust ANOVA indicated that early left AI activity for inclusionary throws during the inclusion phase was similar to activity for exclusionary throws during the inclusion phase, $T_{WJt}/c(1.0,28.0)=3.58$, $p=.07$. For exclusionary throws left AI activity was similar for throws during the inclusion phase and for throws during the first block of the exclusion phase, $T_{WJt}/c(1.0,28.0)=0.67$, $p=.43$.

The second scoring window for left AI corresponded to exclusion SP activity. Left AI activity was marginally greater for inclusionary throws during the inclusion phase than for exclusionary throws during the inclusion phase, $T_{WJt}/c(1.0,28.0)=4.28$, $p=.05$. Inconsistent with exclusion SP findings, the exclusionary throws during the first block of the exclusion phase showed greater activity than the exclusionary throws during the inclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=13.14$, $p=.002$. This latter pattern of effects indicates that late left AI activity was sensitive to the social exclusion manipulation.

Right AI. The first scoring window for right AI corresponded to N2 activity. Unlike the scalp N2 effects, the robust ANOVA for right AI activity for inclusionary and exclusionary throws during the inclusion phase was not significant, $T_{\mathrm{WJt}}/c(1.0,28.0)=3.14$, $p=.09$. Right AI activity was similarly not significant when comparing exclusionary throws during the inclusion phase to exclusionary throws during the first block of the exclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=0.16$, $p=.70$.

The second scoring window for right AI corresponded to exclusion SP activity. Right AI activity was greater for inclusionary throws during the inclusion phase than for exclusionary throws during the inclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=18.83$, $p=.001$. Similar to late left AI activity, late right AI activity was sensitive to the social exclusion manipulation. In the comparison of the exclusionary throws during the inclusion phase to exclusionary throws during the first block of the exclusion phase, greater activity was observed during the exclusion phase than during the inclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=24.31$, $p<.001$.

Right vlPFC. The scoring window for right vlPFC corresponded to exclusion SP activity. Paralleling exclusion SP findings, greater activation was observed during inclusionary throws than during exclusionary throws, as indicated by a robust ANOVA on right vlPFC activity for throws during the inclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=9.62$, $p=.007$. Right vlPFC was also observed to be sensitive to the social exclusion manipulation. Activity for exclusionary throws during the first block of the exclusion phase was greater than activity for exclusionary throws during the inclusion phase, $T_{\mathrm{WJt}}/c(1.0,28.0)=6.80$, $p=.03$.

PCC. The scoring window for PCC corresponded to parietal P3 activity. Consistent with parietal P3 findings, a robust ANOVA for PCC activity during the inclusion phase indicated a nonsignificant difference between inclusionary and exclusionary throws, $T_{WJt}/c(1.0,28.0)=2.99$, $p=.10$. The robust ANOVA for PCC activity for exclusionary throws yielded a nonsignificant effect, PCC activity was similar across the inclusion phase and the first block of the exclusion phase, $T_{WJt}/c(1.0,28.0)=3.95$, $p=.06$. Given the similarity between the pattern of effects for PCC and for parietal P3, these findings implicate PCC in parietal P3 generation.

SFG. The scoring window for SFG also corresponded to parietal P3 activity. A robust ANOVA on SFG activity for throws during the inclusion phase yielded a nonsignificant effect, $T_{WJt}/c(1.0,28.0)=2.14$, $p=.16$. SFG activity for exclusionary throws during the inclusion phase was similar to activity for exclusionary throws during the first block of the exclusion phase, $T_{WJt}/c(1.0,28.0)=1.58$, $p=.22$. This pattern of effects was similar to the pattern observed for parietal P3 and implicates SFG in parietal P3 generation.

Correlational Analyses

Scalp- and source-space amplitudes for exclusionary throws during the first block of the exclusion phase were compared to a self-report measure of social distress, changes in PANAS state negative and positive affect scores, and brief mood scores. None of the correlations was significant for scalp-space analyses ($|r\text{s}| < .27$, $p\text{s} > .15$). This finding is consistent with hypothesis 1b, in that N2 activity was not significantly related to NTS Total scores, changes in PANAS state negative and positive affect scores, or brief mood scores. However, contrary to hypothesis 2c exclusion SP and frontal P3 amplitudes were not significantly correlated with social distress scores.

For source-space analyses, higher NTS Total scores were associated with higher late left AI, late right AI, and right vlPFC activity; *r*(27)=.62, *p*<.001; *r*(27)=.41, *p*=.03; *r*(27)=.40, *p*=.03, respectively. Late bilateral AI activity was more strongly associated with NTS Total scores than early AI activity (|*Z*s| > 3.2, *p*s < .002). Lateralization effects were not observed for late AI activity, *Z*=-1.03, *p*=.30. None of the remaining correlations between source-space amplitudes and NTS Total scores were significant (|*r*s| < .28, *p*s > .14). For the Bad-Good mood score, higher late left AI activity was related to lower scores, which was consistent with bad mood scores, *r*(27)=-.44, *p*=.02. The relationship between late left AI activity and Bad-Good mood scores was stronger than the relationship between early left AI activity and Bad-Good mood scores, *Z*=-2.29, *p*=.02. Lateralization effects were not observed for late AI activity, *Z*=-1.12, *p*=.26. For the Sad-Happy mood score, higher SFG activity was related to lower scores, which were consistent with sad mood scores, *r*(27)=-.38, *p*=.04. None of the remaining correlations with mood item scores or PANAS state negative and positive affect difference scores was significant (|*r*s| < .34, *p*s > .07). Unlike the analyses examining all exclusionary throws during the exclusion phase (see Appendix), late dACC activity for exclusionary throws during the first block of the exclusion phase was not related to any brief mood items.

The relationship between scalp- and source-space activity and levels of social support, as measured by the SSQ, was also examined. Increased late dACC activity for exclusionary throws during the first block of the exclusion phase was associated with a greater number of people who are available to the individual and can be relied on for social support, *r*(29)=.41, *p*=.03. Late dACC activity was more strongly correlated with levels of SSQ social support than early dACC activity was, *Z*=2.58, *p*=.01. The remaining correlations were not significant (|*r*s| < .30, *p*s > .11). Contrary to hypothesis 2e, exclusion SP and frontal P3 were not significantly correlated with levels of social support, and greater levels of social support were related to increased, rather than decreased, dACC activity.

Exploratory Analyses

dACC Activity During the Exclusion SP Time Window. In previous source localization work, exclusion SP was observed to be a partial manifestation of dACC activity (Crowley et al., 2010). This possibility was explored for the present data. The temporal window corresponding to exclusion SP (600 – 900 ms) was scored for dACC activity and subsequently analyzed. Contrary to the pattern observed for exclusion SP, a robust ANOVA on dACC activity corresponding to the exclusion SP temporal window indicated similar levels of activity for inclusionary throws and exclusionary throws during the inclusion phase, $T_{WJt}/c(1.0,28.0)=0.52$, $p=.47$. Similar levels of activity were also observed for exclusionary throws during the inclusion phase and for exclusionary throws during the first block of the exclusion phase, $T_{WJt}/c(1.0,28.0)=1.01$, $p=.33$. This latter analysis comparing activity between inclusion and exclusion phases is consistent with findings above for early and late dACC activity. Taken together, dACC activity for exclusionary throws did not significantly differentiate between the inclusion phase and the first block of the exclusion phase in any of the temporal windows examined. None of the correlations was significant between dACC activity corresponding to the exclusion SP temporal window and a self-report measure of social distress, changes in PANAS state negative and positive affect scores, or brief mood scores ($|r\text{s}| < .23$, $p\text{s} > .19$).

Scalp- and Source-Level Activity Correlations. In an effort to evaluate whether the regional sources account for the scalp activity of interest, scalp-level component amplitude was correlated with regional source waveform activity (see Table 5). For these correlations, each scalp-level component was correlated with the regional source activity that corresponded to the temporal window of the scalp-level activity. For example, N2 amplitude was correlated with early dACC activity, early left AI activity, and early right AI activity but not with right vlPFC activity, PCC activity, or SFG activity, as the temporal windows used for scoring these regional sources were later than the N2 time window.

Consistent with hypothesis 1c, early dACC activity correlated with N2 amplitude for exclusionary throws during both phases, but not for inclusionary throws during the inclusion phase. Consistent with hypothesis 2d, late dACC activity was correlated with frontal P3 amplitude for exclusionary throws during the inclusion phase, and dACC activity was also correlated with parietal P3 amplitude for exclusionary throws during both phases but not for inclusionary throws during the inclusion phase. In harmony with a previous exclusion SP source localization study (Crowley et al., 2010), dACC activity corresponding to the exclusion SP temporal window was correlated with exclusion SP for exclusionary throws during the inclusion phase.

Consistent with some source localization work suggesting a role of AI in conflict-related N2 generation (e.g., Menon, Adleman, White, Glover, & Reiss, 2001; Rubia et al., 2001), early left AI activity was correlated with N2 amplitude for exclusionary throws during the exclusion phase. However, the remaining correlations between early left and right AI activity and N2 amplitude were not significant. Lateralization effects for early AI activity and N2 amplitude were not observed for any event types ($|Z\text{s}| < 0.9$, $p\text{s} > .39$). Contrary to expectations based on previous ERP source localization with the Cyberball task (Crowley et al., 2010), late left and right AI activity were not significantly correlated with exclusion SP amplitude. Lateralization effects for late AI activity and exclusion SP were not observed for any event types ($|Z\text{s}| < 1.1$, $p\text{s} > .30$). Right vlPFC activity was also not correlated with exclusion SP amplitude.

PCC activity was significantly correlated with frontal and parietal P3 amplitudes for most event types. Although a prior Cyberball ERP study implicated PCC in exclusion SP generation (Crowley et al., 2010), significant correlations between PCC activity during the exclusion SP time window and exclusion SP were not observed. Consistent with some prior P3b work (e.g., Galashan et al., 2015), SFG activity was significantly correlated with parietal P3 activity for inclusionary throws during the inclusion phase and for exclusionary throws during the exclusion phase. SFG activity was also marginally significantly related to frontal P3 amplitude for exclusionary throws during the inclusion phase.

Discussion

The present examination sought to evaluate the functional significance of early and late exclusion-related source and scalp activity by using a modified Cyberball paradigm and to determine the extent to which dACC activation observed during the exclusion phase of the Cyberball was related to violating an expectancy of inclusion, social distress and negative affect, or a combination of both. Additionally, this study laid the groundwork for using a modified Cyberball paradigm to examine social exclusion in schizophrenia in an effort to understand how sensitivity to social exclusion impacts symptoms and relapse in patients. The functional significance of dACC activation during the Cyberball task remains a matter of considerable controversy (for review, see Eisenberger, 2015). Interpretations of the role of dACC have relied on a large body of fMRI research and attempted to conclude that dACC is involved in either expectancy violations or social distress and negative affect. The present findings suggest that previous fMRI research attempting to determine the functional significance of dACC during the Cyberball task may have been limited by the low temporal resolution of fMRI. This study capitalized on the high temporal resolution of EEG by parsing dACC activity into early and late temporal windows. Current analyses provide some evidence that early dACC activity is sensitive to violations of expectancy and late dACC activity is sensitive to changes in negative affect. This is the first study to explicitly examine the neural time course of dACC activity during the Cyberball task, benefiting from the temporal resolution of EEG, and suggests that the functional significance of dACC is not fully accounted for by either expectancy violations or social distress and negative affect alone.

Social Exclusion Manipulation

On the basis of self report, the social exclusion manipulation appeared successful. Participants endorsed moderate levels of NTS social distress and decreases in PANAS state positive affect following the social exclusion phase. Participants also reported levels of NTS social distress similar to those reported in previous studies (Eisenberger et al., 2006; Masten et al., 2011; Xu et al., 2016) and reported being moderately ignored and excluded. Consistent with previous findings (e.g., Seidel et al., 2013; Themanson et al., 2015), PANAS state positive affect decreased from pre-task to post-task measurements. However, PANAS state negative affect scores also decreased. Previous Cyberball research has generally observed PANAS state negative affect scores increasing from baseline (Dixon-Gordon, Gratz, Breetz, & Tull, 2013; Themanson et al., 2015; Zhang et al., 2017) or remaining similar to baseline (Seidel et al., 2013; Themanson et al., 2013; Xu et al., 2016) after the social exclusion phase. The combination of low negative affect and low positive affect in the present study may reflect an absence of affective involvement or a lack of task engagement (Russell & Carroll, 1999; Watson & Tellegen, 1985). Such a possibility is consistent with the neutral responses to the brief mood items (e.g., Bad/Good and Sad/Happy). A possible explanation for an apparent disengagement or absence of affective involvement after the Cyberball task may be that participants were tired or unmotivated following the high number of trials during the exclusion phase.

Given that the pattern of effects for the scalp- and source-space analyses indicated a decrease in activity for most measures, task disengagement may account for the some of the decreased neural activity in the analyses using all exclusionary throws during the exclusion phase. For example, P3a and P3b (frontal and parietal P3, respectively) have been observed to be positively correlated with estimates of task engagement (Boksem, Meijman, & Lorist, 2006; Hopstaken, van der Linden, Bakker, & Kompier, 2015; Murphy, Robertson, Balsters, & O'Connell, 2011). Considerable evidence also indicates that dACC activity increases in conjunction with increased task engagement (Botvinick, 2007; Duncan, 2010; Nachev, Wydell, Neill, Husain, & Kennard, 2007; Paus, Koski, Caramanos, & Westbury, 1998; Venkatraman & Huettel, 2012). A decrease in frontal P3, parietal P3, and dACC activity being associated with a decrease in task engagement is consistent with an interpretation that task engagement decreased from the first to the second block of the exclusion phase.

Although a high number of exclusionary throws were needed to obtain adequate signal-to-noise ratios to construct source models, including one inclusionary throw in each ten-trial set of throws during the exclusion phase did not appear to sufficiently maintain task engagement throughout the exclusion phase. A recent meta-analysis of Cyberball studies speculated that increasing the number of throws may be associated with a diffused ostracism effect (Hartgerink, van Beest, Wicherts, & Williams, 2015), which could have decreased the potency of the social exclusion manipulation despite similar levels of social distress to previous studies. In light of the abovementioned findings, subsequent discussion largely emphasizes the findings during the first block of the exclusion phase, when participants were likely more engaged in the task and when the social exclusion manipulation was likely more salient.

Early Exclusion-Related Activity

During the Cyberball task, dACC activation during the exclusion phase has been attributed to an expectancy violation, social distress and negative affect, or a combination of both (e.g., Eisenberger, 2015). In fMRI studies, greater dACC activation has generally been observed during the exclusion phase than during the inclusion phase, and dACC activation during the exclusion phase was associated with self-report measures of social distress (e.g., Eisenberger et al., 2003; Masten et al., 2009). However, previous ERP studies examining N2, a putative manifestation of early dACC activity (van Veen & Carter, 2002; Yeung et al., 2004), observed similar N2 amplitudes in inclusion and exclusion phases for exclusionary throws and a nonsignificant relationship between N2 amplitudes for exclusionary throws during the exclusion phase and self-report measures of social distress (Themanson et al., 2013; Themanson et al., 2015). That is, whereas fMRI studies observe dACC activation to be associated with changes in self-reported social distress, N2 findings suggest that early dACC activity is related to violations of expectancy.

Similar to previous ERP studies (Gutz et al., 2011; Themanson et al., 2013; Themanson et al., 2015), the present examination provides some support for the notion that N2 and early dACC activity are sensitive to expectancy violations rather to changes in social distress. Greater early dACC activity was observed to exclusionary throws than to inclusionary throws during the inclusion phase, which is a violation of the expectancy to be included during the inclusion phase. Although N2 amplitudes were larger to inclusionary throws than to exclusionary throws during the inclusion phase, overlapping EEG activity, such as frontal or parietal P3, may have accounted for the larger N2 to inclusionary throws than to exclusionary throws. If N2 and early dACC activity were sensitive to changes in social distress, activity would be expected to be larger during the social exclusion phase. However, the levels of activity for N2 and early dACC for exclusionary throws during the inclusion phase were similar to exclusionary throws during the first block of the exclusion phase, suggesting that the social exclusion manipulation did significantly not impact N2 or dACC activity. N2 and early dACC activity were also not significantly related to NTS social distress scores, changes in PANAS state negative or positive affect, or the brief mood item scores. These findings suggest that N2 and early dACC activity are sensitive to expectancy violations but not sensitive to changes in social distress or mood scores.

Early dACC activity to exclusionary throws from both phases and early left AI activity to exclusionary throws during the exclusion phase were significantly correlated with N2 activity, suggesting that dACC and/or left AI contributed to N2 activity observed during this Cyberball task. These findings are consistent with other work demonstrating N2 activity in paradigms involving discrepancy detection to be source localized to dACC (e.g., van Veen & Carter, 2002; Yeung et al., 2004) and left AI (e.g., Menon et al., 2001; Rubia et al., 2001). Paralleling N2 findings, early AI activity was similar to exclusionary throws during the inclusion phase and exclusionary throws during the first block of the exclusion phase. However, unlike N2 early AI activity did not significantly differentiate between inclusionary and exclusionary throws during the inclusion phase. Thus, contrary to early dACC activity, early AI activity did not follow the same pattern of effects as N2.

Unlike N2 and early dACC activity, early left and right AI activity showed some relationships to affective ratings from the data examining all exclusionary throws during the exclusion phase (see Appendix). Greater decreases in PANAS state negative and positive affect after the exclusion phase were related to more early left and right AI activity. Cyberball research with fMRI occasionally reports a relationship between AI and measures of social distress (e.g., DeWall et al., 2012; Masten et al., 2012). Present findings documented this relationship for late AI activity, although NTS social distress was not significantly related to early AI activity. This relationship between early AI activity and PANAS scores is consistent with research observing AI activation to be related to various measures of negative affective state (e.g., Lamm & Singer, 2010; Phan, Wager, Taylor, & Liberzon, 2004). Thus, although AI activity appeared to partially contribute to scalp N2 activity, AI activity appears functionally related to changes in affective state following social exclusion.

Although previous fMRI Cyberball research has not parsed dACC activation into early and late activation during exclusionary throws of the exclusion phase, present findings provide some evidence that early dACC activity may not be sensitive to the social exclusion manipulation in the Cyberball task or to self-reported social distress. Instead, the functional significance of early dACC exclusion-related activity during this modified Cyberball paradigm appears to be related to monitoring for expectancy violations.

Late Exclusion-Related Activity

Frontal P3. P3a is sensitive to novel stimuli and then habituates to repeated presentations (Donchin & Coles, 1988; Polich, 2007; Simons et al., 2001), and the present frontal P3 findings appear similar in latency and topography to P3a. The present frontal P3 analyses suggest habituation similar to that for inclusionary and exclusionary throws during the inclusion phase and similar to that for exclusionary throws during the inclusion phase and for exclusionary throws during the first block of the exclusion phase. The sensitivity of frontal P3 to habituation is observed in the decrease from the first block to the second block of throws in the exclusion phase. The high proportion of exclusionary throws was likely more novel during the first block of the exclusion phase. Then, over the course of the exclusion phase there was habituation to the high proportion of exclusionary throws. The functional significance of frontal P3 during the Cyberball paradigm appears to be related to novelty and habituation, which is consistent with a large body of research, rather than to expectancy violations or changes in social distress.

Contrary to present hypotheses, frontal P3 amplitude for exclusionary throws during the first block of the exclusion phase was similar to frontal P3 amplitude for exclusionary throws during the inclusion phase. Previous findings with regard to frontal P3 have been mixed with one study showing larger frontal P3 during the inclusion phase than during the exclusion phase in children (Crowley et al., 2010) and the other study observing the opposite pattern with larger frontal P3 during the exclusion phase than during the inclusion phase in young adults (Themanson et al., 2015). Although frontal P3 amplitude was previously observed to be correlated with PANAS state negative affect (Themanson et al., 2015), frontal P3 amplitude was not significantly correlated with the same measure in the present study, nor with PANAS state positive affect, NTS social distress scores, or mood item scores.

Parietal P3. The scalp topography of parietal P3 appears similar to the scalp topography of a conventional P3b, but the latency of parietal P3 is earlier than the latency of frontal P3, which is inconsistent with previous studies of P3a and P3b. Theoretical explanations of P3a and P3b associate P3a with processes related to early focal attention, which facilitates context-updating and subsequent memory storage putatively reflected in P3b (Donchin, 1981; Hartikainen & Knight, 2003; Kok, 2001; Polich, 2007, 2012). The present findings of parietal P3 preceding frontal P3 are not compatible with these proposed explanations of the functional significance of P3a and P3b.

Previous studies of P3b generally observe that P3b amplitude inversely relates to the probability of a target stimulus (Duncan-Johnson & Donchin, 1977; Squires, Wickens, Squires, & Donchin, 1976). Based on this relationship between stimulus probability and P3b amplitude, parietal P3 amplitude would not be expected to differentiate between inclusionary and exclusionary throws during the inclusion phase. Additionally, exclusionary throws were presented with a high probability during the exclusion phase, which would lead to a small parietal P3. It seems that the stimulus probabilities used in the present paradigm were not appropriately calibrated to elicit a robust parietal P3. Previous studies examining the impact of overinclusion on P3b, observed larger P3b for inclusionary throws during an inclusion phase when participants received 33% of ball throws than for inclusionary throws during an overinclusion phase when participants received 46% of ball throws (Niedeggen, Sarauli, Cacciola, & Weschke, 2014). Future attempts to elicit P3b during the Cyberball paradigm may consider manipulating the probability of throws during the inclusion phase to elicit a robust P3b. Additionally, it would be expected that during the exclusion phase, parietal P3 would be larger to inclusionary throws than to exclusionary throws, given the low probability of inclusionary throws during exclusion. However, adequate reliability for parietal P3 scores for inclusionary throws during the exclusion phase was not obtained in the present dataset. Future research interested in parietal P3 during the exclusion phase may consider using longer exclusion phases to obtain the high number inclusionary throws necessary for adequate parietal P3 score reliability.

These limitations notwithstanding, parietal P3 findings paralleled those of frontal P3. Previous P3b findings have been mixed, with one study observing similar amplitudes for exclusionary throws during the inclusion and exclusion phases (Themanson et al., 2015) and the other study observing larger P3b to exclusionary throws during the exclusion phase than to exclusionary throws during the inclusion phase (Themanson et al., 2013). Present parietal P3 findings were consistent with the former study. Additionally, the decrease in parietal P3 from the first block to the second block of the exclusion phase seems consistent with parietal P3 being sensitive to context-updating, given the reduced novelty and repetitive nature of the exclusion phase. However, this interpretation is purely speculative in light of the limitations described above.

dACC, PCC, and SFG. Based on findings from the source model, dACC, PCC, and SFG regional sources appear to account for at least some of the activity contributing to frontal and parietal P3. These findings are in agreement with source localization studies implicating dACC, PCC, and/or SFG in P3a/P3b generation (e.g., Crottaz-Herbette & Menon, 2006; Galashan et al., 2015). Similarly, fMRI studies have observed activation of dACC and/or PCC during the Cyberball task (e.g., Eisenberger et al., 2003; Onoda et al., 2009), but activation of SFG is less commonly observed (cf. Gonzalez et al., 2015). Contrary to previously cited research, none of these three regional sources showed greater activity for exclusionary throws during the first block of the exclusion phase than for exclusionary throws during the inclusion phase.

Present findings for the analyses examining all exclusionary throws during the exclusion phase provide some evidence that late dACC activity is more strongly related to changes in self-reported mood scores than early dACC activity. Late dACC activity from the analyses examining all exclusionary throws from the exclusion phase was more strongly related to increased sad and unfriendly mood scores than early dACC activity (see Appendix). However, significant relationships between late dACC activity during the first block of the exclusion phase and mood items scores were not observed, and late dACC activity was also not related to changes in

PANAS state negative affect scores. Contrary to hypotheses, late dACC activity was not significantly related to NTS social distress. This finding stands in contrast to a large body of fMRI literature that commonly observes a positive association between dACC activation and self-report measures of social distress and greater dACC activation during social exclusion than during social inclusion (for review, see Eisenberger, 2012).

Limitations of EEG source localization notwithstanding, a number of factors may have contributed to the nonsignificant correlation between dACC activity and NTS social distress. One possibility for the nonsignificant finding is how temporally removed the NTS administration was from the first block of the exclusion phase when the social exclusion was likely more salient due to the high number of trials included in the social exclusion phase. Over the course of the exclusion phase, the NTS scores may have been impacted by task disengagement, which obscured the relationship between dACC activity and self-reported social distress. However, since dACC activity was similar for exclusionary throws between the inclusion phase and first block of the exclusion phase, it is unclear whether assessing social distress immediately following the first block of the exclusion phase would have yielded strikingly different findings, as fMRI Cyberball studies consistently yield larger dACC activity during the exclusion phase than during the inclusion phase.

Another possible explanation for why dACC activity was not correlated with NTS social distress scores was that the present paradigm deviated from the common implementation of the Cyberball task. The present modified Cyberball paradigm used partial social exclusion, rather than full social exclusion, as is commonly used in the fMRI implementation of the Cyberball task. Partial exclusion may represent a qualitatively different type of stressor and may not have been potent enough as a social exclusion manipulation to elicit adequate levels of social distress, although present levels of self-reported NTS social distress were similar to those reported in other Cyberball studies (Eisenberger et al., 2006; Masten et al., 2011; Xu et al., 2016).

With regard to the functional significance of dACC activation during the Cyberball task, present findings are consistent with prevailing theoretical explanations that dACC is involved in responding to expectancy violations and to pain and negative affect (Eisenberger, 2015; Eisenberger & Lieberman, 2004; Spunt et al., 2012). These explanations posit that dACC first detects an expectancy violation and then is associated with pain and negative affect. The current findings bolster this work by observing this temporal distinction between early and late dACC activity. Given this temporal distinction, future attempts to determine the role of dACC during social exclusion may consider the neural time course of dACC functioning when evaluating its functional significance.

Exclusion SP. Contrary to predictions, exclusion SP amplitude did not differentiate between exclusionary throws during the inclusion and exclusion phases and was unrelated to measures of social distress. In previous research, exclusion SP differentiated between exclusionary throws during the inclusion and exclusion phases, and increased exclusion SP was related to increased self-reported social distress in children (Crowley et al., 2010; McPartland et al., 2011) and young adults (Crowley et al., 2009). There are substantial paradigmatic differences between the present Cyberball paradigm and the paradigm used in previous studies of the exclusion SP. For example, in the previous exclusion SP studies when the ball was passed during the game, sound effects occurred when the ball traveled and when it landed in the receiving baseball mitt. The ball also followed a straight line, arc, or sine wave when passed. Those two features prove particularly troublesome for ERP analysis, as the inclusion of sound produces auditory ERP signals and requiring participants to visually track the ball produces ocular artifact. The inclusion of motion is especially problematic as the ocular artifact in the EEG is much larger than ERP components of interest. It is possible that these differences could have made the task more engaging than the present modified paradigm. However, by eliciting auditory ERPs and ocular artifact these paradigmatic differences likely produced noise in the EEG recordings that overlapped with ERP signals of interest, thus reducing the signal-to-noise ratio of the averaged ERPs and possibly impacting exclusion SP scores. It is also possible that the use of sound and/or requiring the participants to visually track the ball are necessary for eliciting exclusion SP. Additional research examining the necessary paradigmatic features that lead to exclusion SP is needed before it is possible to determine its functional significance.

AI and vlPFC. Although a correlation between exclusion SP and scores for late AI and right vlPFC activity were not significantly associated, these regional sources showed the most consistent pattern of greater activation during the first block of the exclusion phase and relationships with self-report measures of social distress. Bilateral AI activity and right vlPFC activity were greater during the first block of the exclusion phase than during the inclusion phase. AI activation is observed to be larger during the social exclusion phase than during the inclusion phase in fMRI research (Dewall et al., 2010; Eisenberger et al., 2003; Masten et al., 2011; Masten et al., 2012). Higher NTS social distress scores were associated with higher late AI and right vlPFC activity for scores from all exclusionary throws during the exclusion phase as well as for scores from exclusionary throws during the first block of the exclusion phase. A fairly consistent finding in the Cyberball literature is a positive association between left, right, or bilateral AI activation and self-reported social distress (DeWall et al., 2012; Masten et al., 2009; Masten et al., 2011; Masten et al., 2012). Present findings are consistent with the notion that AI activity is sensitive to the social exclusion manipulation and is associated with changes in social distress measures following social exclusion. These findings are compatible with previous suggestions that AI responds to the affective component of social pain following social exclusion (Eisenberger, 2012, 2015).

In summary, late exclusion-related activity showed more consistent relationships with self-reported social distress than early exclusion-related activity, and late dACC activity showed more associations with mood scores than early dACC activity. Additionally, late exclusion-related activity as measured by bilateral AI and right vlPFC showed greater activity during the first block of the exclusion phase than during the inclusion phase, suggesting that these brain regions were sensitive to the social exclusion manipulation. However, present findings should be interpreted in light of the failure to replicate some findings from the Cyberball literature. A number of potential explanations are proposed, such as the use of partial social exclusion rather than full social exclusion. Even after findings from the first block of the exclusion phase were examined when task engagement was likely high and the social exclusion manipulation was likely salient, the present analyses failed to replicate the commonly observed pattern of greater dACC activity during social exclusion than during social inclusion and the relationship between dACC activity and NTS social distress (for reviews, see Eisenberger, 2012; Rotge et al., 2014). Contrasting ERP and fMRI approaches to examining data obtained during the Cyberball may partially explain some of these discrepant findings.

Contrasting ERP and fMRI Cyberball Research

As previously noted, the use of partial exclusion and the high number of throws included in the present version of the Cyberball task represent a clear difference from the Cyberball procedures commonly used in fMRI research. A meta-analysis of fMRI Cyberball studies indicated that the duration of the social exclusion phase generally lasts between 20 and 120 seconds, with most studies using exclusion durations under 80 seconds (Rotge et al., 2014). The present paradigm used 160 trials for the exclusion phase that lasted approximately six minutes, and the participant received the ball once per every set of 10 trials. Although it has been speculated that increasing the length of the exclusion phase may result in a diffused ostracism effect (Hartgerink et al., 2015), the present version of the Cyberball resulted in levels of NTS social distress similar to previous studies (see Eisenberger et al., 2006; Masten et al., 2011; Xu et al., 2016), suggesting that the social exclusion manipulation was comparable in effectiveness to previous research. Although the use of partial exclusion and the high number of trials represent a paradigmatic deviation from fMRI studies using the Cyberball task, these changes were necessary to obtain an adequate signal-to-noise ratio for source modeling.

In an attempt to circumvent the limitation of including a high number of trials, analyses were conducted on the first 20 exclusionary throws of the exclusion phase. Meta-analytic work indicates that shorter durations of exclusion were associated with greater ventral ACC activation than longer durations of exclusion, which were associated with greater dACC activation (Rotge et al., 2014). In the present study, dACC activity was similar across blocks of the exclusion phase, suggesting that the duration of the exclusion phase did not impact dACC activity. However, it is possible that ACC exclusion-related activation during the first block of the exclusion phase was not optimally modelled by a dACC regional source, which represents another difference between the present study and some previous fMRI investigations.

Previous fMRI research largely examined differences using a blocked design (cf. Kawamoto et al., 2012), comparing signal between the inclusion phase and the exclusion phase, but the present ERP analyses used an event-related design to compare activity between exclusionary throws from each phase. That is, fMRI analyses contrast brain activity for inclusionary and exclusionary throws during the inclusion phase to activity for exclusionary throws during the exclusion phase. Unlike an event-related design, a block design results in the inclusion of activity to the trials and inter-trial intervals. Additionally, blood oxygen level-dependent (BOLD) fMRI signal change represents an indirect measure of neural activity, and its time course is somewhat sluggish, reaching peak activation 4 to 6 seconds after change in neural activity (Aguirre, Zarahn, & D'Esposito, 1998; Bandettini, Wong, Hinks, Tikofsky, & Hyde, 1992; Boynton, Engel, Glover, & Heeger, 1996). Exclusion-related activation of brain regions may occur across trials and their inter-trial intervals during the social exclusion phase and consequently was not captured by EEG (cf. Crowley et al., 2010).

Evaluating the EEG Source Model

The current EEG source model used candidate brain regions from fMRI Cyberball research (Eisenberger et al., 2003; Masten et al., 2009; Onoda et al., 2009) and P3a/P3b source localization studies (Crottaz-Herbette & Menon, 2006; Galashan et al., 2015; Kiehl et al., 2001). In addition to the six regional sources of interest, three additional regional sources were used to capture noise, including activity outside the candidate brain regions, residual ocular artifact, and background EEG noise. Based on the pattern of findings between source and scalp activity as well as correlations between source and scalp activity, the source solution appeared to adequately model N2, frontal P3, and parietal P3 activity. dACC activity during the exclusion SP temporal window significantly correlated with exclusion SP amplitude for one event type, but dACC activity did not show a similar pattern of effects as exclusion SP. PCC activity during the exclusion SP time window was also unrelated to exclusion SP (cf. Crowley et al., 2010). Although late left and right AI and right vlPFC activity showed fairly similar patterns of activity to exclusion SP, correlations between source activity for these regions and exclusion SP were not observed, suggesting that the neural generators of exclusion SP were not adequately modeled.

A previous source localization study of exclusion SP in children implicated regions including subgenual cingulate cortex, ventral posterior cortex, insula, ACC, and posterior cingulate cortex (Crowley et al., 2010). Considering that exclusion-related activity may manifest differently across development (Rotge et al., 2014) and that not all candidate regions were modeled due to limitations of discrete source modeling, key regions in exclusion SP generation may have been missed in the current source model. One pertinent limitation of Crowley et al. (2010) may have been the use of a regression-based ocular artifact correction procedure prior to EEG source analysis. Artifact and brain topographies, particularly for the frontally-distributed exclusion SP, are spatially correlated, and the use of a regression-based correction procedure for removing ocular artifact severely distorts the scalp EEG data and the resulting source localization (Berg & Scherg, 1994b; Huotilainen et al., 1995; Scherg, Ille, Bornfleth, & Berg, 2002). The present study employed an adaptive artifact correction approach that used ocular artifact templates tailored to each individual subject's data (Ille et al., 2002). The adaptive artifact correction approach considers both the artifact signals and brain activity during source analyses. By modeling artifact signals and brain activity, ocular artifact can be removed without distorting the brain activity even when the artifact signals and brain activity are spatially correlated, as is the case with exclusion SP. Additionally, a regional source was included in the source model to account for residual ocular artifact during source analysis to further reduce the potential impact of ocular activity on the regional sources of interest. Lastly, the present study had the comparative advantage of digitizing the individual position of each scalp sensor for each subject, which can improve source localization of scalp activity (Akalin Acar & Makeig, 2013;

Beltrachini, von Ellenrieder, & Muravchik, 2011; Khosla, Don, & Kwong, 1999; Koessler et al., 2007; Wang & Gotman, 2001). Thus, the present source model likely provides an acceptable estimate of brain activity, despite deviations between present findings and a previous ERP Cyberball source localization study (Crowley et al., 2010).

A possible criticism of the present source model was that a key brain region, dACC, failed to show the common pattern of greater activity during the social exclusion phase than during the social inclusion phase. However, activity in other brain regions was consistent with prior fMRI work. For example, AI activity for exclusionary throws was greater during the first block of the exclusion phase than during the inclusion phase (e.g., Dewall et al., 2010; Masten et al., 2011) and correlated with NTS social distress scores, which is a commonly observed relationship in the fMRI literature (e.g., DeWall et al., 2012; Masten et al., 2012). This relationship provides some evidence that AI activity was adequately modeled by showing congruence with fMRI findings. Furthermore, scalp activity did not clearly differentiate between the social inclusion phase and the first block of the social exclusion phase and significant correlations between NTS social distress scores and scalp activity were not observed, suggesting that modeling source activity provided incremental value beyond examining activity at the scalp alone. The nonsignificant differentiation between social inclusion and exclusion phases for dACC activity does not seem to be a weakness of the source model per se. Rather, the nonsignificant differentiation between social inclusion and exclusion phases for dACC activity appears to be a weakness of the present Cyberball paradigm.

Considering the paradigmatic differences between this modified Cyberball paradigm and those used in fMRI research highlighted above, it is possible that the regional sources were not precisely seeded. A regional source models diffuse activity in several square centimeters of proximal brain tissue (Paul-Jordanov et al., 2016). Another possibility for the nonsignificant differentiation between social inclusion and exclusion phases is that exclusion-related activity was contaminated by surrounding brain activity. For example, a meta-analysis of fMRI studies investigating the impact of social exclusion on ACC activation indicated patterns of BOLD signal in various patches of ventral ACC and dACC (Rotge et al., 2014). It is possible that the spatial location of the dACC regional source in the present study only partially accounted for activity sensitive to social exclusion. Future research aimed at identifying the neural time course of dACC activity during the Cyberball task would benefit from the use of simultaneously recorded EEG and fMRI, so that regional sources can be seeded based on candidate brain regions identified via fMRI. Additionally, information from the structural MRIs could be used to constrain the solution space to the brain anatomy of individual subjects in an effort to improve the accuracy of source localization.

Additional Limitations

A number of potential limitations have been highlighted, including the length of the exclusion phase, the use of partial exclusion, and paradigmatic differences between the present paradigm and previous Cyberball tasks. An additional limitation of the present study is that it may have been underpowered to detect significant differences between inclusion and exclusion phases or correlations between neural activity and self-report measures. Of the 26 fMRI Cyberball studies reported in a recent meta-analysis (Rotge et al., 2014), the range of sample sizes was 10 to 53 ($M = 24$, $SD = 9$). For ERP studies (Crowley et al., 2009; Crowley et al., 2010; Gutz et al., 2011; McPartland et al., 2011; Themanson et al., 2013; Themanson et al., 2015), the range of sample sizes was 20 to 55 ($M = 31$, $SD = 11$). Depending on the analyses, the present study used data from 29 to 32 participants, which is greater than the average number of participants in fMRI studies and similar to the average number of participants for ERP studies. Considering that the paradigmatic differences between this study and previous Cyberball studies may have reduced the impact of the social exclusion manipulation, it is possible that the present study was underpowered to detect differences between social inclusion and exclusion phases for source- and scalp-activity. Consequently, nonsignificant differentiation between social inclusion and exclusion phases should be interpreted cautiously.

Contrary to prior Cyberball studies, the present study introduced participants to a confederate and participants viewed photographs of all three players on the computer screen prior to and after the Cyberball task. This was done in an effort to improve the believability and potency of the social exclusion manipulation. As previously discussed, the present modified Cyberball paradigm also avoided the use of visual motion and ball throws in an effort to avoid unnecessary contamination of EEG signal with excessive eye movement. Instead, the modified Cyberball paradigm used an arrow to indicate the direction of the ball throw and then the ball appeared next to the stationary baseball mitt of the receiving player. Such paradigmatic differences could have resulted in decreased task engagement or had an unexpected impact on neural activity that influenced the present findings.

Conclusions and Future Directions

The present study sought to develop a version of the Cyberball task optimized for ERP recordings in an effort to functionally dissociate early and late exclusion-related scalp and source activity, particularly for dACC. Present findings provide some evidence for early dACC activity being sensitive to exclusion-related activity and late dACC activity being sensitive to changes in mood scores. However, early and late dACC activity did not significantly differentiate between social inclusion and exclusion phases. Additionally, late dACC was not significantly related to

NTS social distress, which was contrary to present hypotheses. This nonsignificant relationship for late dACC activity stands in contrast to a large body of fMRI literature examining social distress with the Cyberball paradigm (for review, see Eisenberger, 2012).

Although frontal P3, parietal P3, and exclusion SP failed to differentiate between inclusion and exclusion phases and failed to show relationships to measures of social distress, late source activity differentiated between the phases and showed some relationships to social distress measures. Findings for late source activity, such as bilateral AI and right vlPFC, were fairly congruent with fMRI research (for review, see Eisenberger, 2012). These findings suggest that there is value in modelling ERP source activity during the Cyberball task to examine differences between social inclusion and exclusion phases and to investigate relationships between source activity and self-report measures.

The current examination provides an important step toward optimizing the Cyberball paradigm for ERP studies. However, significant obstacles remain before this modified Cyberball task can be used to study patients with schizophrenia. To potentially replicate fMRI Cyberball research, future implementations of an ERP Cyberball paradigm might consider the use of full rather than partial exclusion. Similar to the long exclusion phase used in the present study, previous ERP Cyberball studies also relied on substantially longer exclusion phases than the majority of fMRI research in order to achieve reliable ERP measurements. However, none of the previous ERP studies examined the reliability of the obtained ERP scores. Generalizability of present score reliability to future studies of healthy participants or patients with schizophrenia notwithstanding (see Clayson & Miller, 2017b), the present data suggest that reliable ERP scores are observed with a short exclusion phase similar to fMRI studies. Although the tradeoff of this approach is a reduction in the signal-to-noise ratio for source analyses during the social exclusion phase, there may be an increase in the potency of the social exclusion manipulation that is observable in scalp ERP activity (e.g., Themanson et al., 2015). EEG source analysis would be improved by using individual structural MRIs to constrain sources to individual-specific brain anatomy and by concurrently recording with fMRI to guide the placement of regional sources. Incorporating such approaches would improve the modeling of the neural time course of ACC activity in an effort to further determine whether early or late activity is accounted for by expectancy violations, social distress and negative affect, or a combination of the two.

Although findings from the present study diverged somewhat from previous fMRI and ERP Cyberball research, encouraging and important directions for future research remain. Continued optimization of the Cyberball paradigm for the recording of ERPs will hopefully resolve the uncertainties and inconsistencies observed thus far in the ERP Cyberball literature and shed light on the functional dissociation between early and late exclusion-related activity. Once established for the recording of ERPs, the paradigm may prove to be quite useful for studying the impact of social exclusion as a type of SET in patients with schizophrenia. Such research may identify a plausible mechanism by which sensitivity to social exclusion impacts symptoms and relapse in patients with schizophrenia.

Tables

Table 1

Dependability Summary Information for Event-Related Potentials (ERPs) as a Function of Event Type and Component

Component	Phase	Throw	Trial Cutoff	Overall Dependability
N2	Inclusion	Inclusionary	14	.89 (.84, .94)
		Exclusionary	11	.92 (.87, .95)
	Exclusion	Exclusionary	7	.97 (.96, .98)
Frontal P3	Inclusion	Inclusionary	20	.85 (.77, .92)
		Exclusionary	22	.84 (.75, .91)
	Exclusion	Exclusionary	13	.95 (.92, .97)
Parietal P3	Inclusion	Inclusionary	18	.86 (.79, .92)
		Exclusionary	16	.88 (.82, .93)
	Exclusion	Exclusionary	12	.95 (.92, .97)
Exclusion SP	Inclusion	Inclusionary	30	.80 (.68, .89)
		Exclusionary	26	.82 (.72, .90)
	Exclusion	Exclusionary	7	.97 (.96, .98)

Note. Trial cutoffs represent the number of trials needed to obtain a dependability estimate of .70 for a given ERP component and event type. The overall dependability represents the dependability point estimates and their 95% credible intervals for data including all trials (*n* = 32). Exclusion SP = exclusion slow potential

Table 2

Montreal Neurological Institute (MNI) Coordinates and Temporal Windows Used for Fitting the Regional Sources

Region	MNI Coordinates (x, y, z)	Temporal Window (ms)
dACC	(-8, 20, 40)	200 – 445
Left AI	(-46, 8, -4)	600 – 700
Right AI	(46, 8, -4)	600 – 700
Right vlPFC	(52, 34, 8)	600 – 700
Right PCC	(3, -38, 46)	300 – 400
Right SFG	(3, 2, 59)	300 – 400
Ocular Noise Source	(0, 68, -29)	--
Left Occipital Cortex Noise Source	(-24, -86, -9)	--
Right Occipital Cortex Noise Source	(24, -86, -9)	--

Note. Temporal window refers to the timeframe used for orienting one dipole of the regional source. dACC = dorsal anterior cingulate cortex, AI = anterior insula, vlPFC = ventrolateral prefrontal cortex, PCC = posterior cingulate cortex, SFG = superior frontal gyrus

Table 3

Mean Summary Data for the Manipulation Checks

	M	*SD*	Range	Cronbach's α
NTS – Belongingness[1]	2.5	0.9	1 to 4.3	.83
NTS – Control[1]	4.5	0.5	3 to 5	.87
NTS – Meaningful Existence[1]	2.1	1.0	1 to 4	.90
NTS – Self-Esteem[1]	3.7	0.7	2.3 to 5	.82
NTS – Average Score	3.2	0.5	2.4 to 4.2	.80
Percentage of Throws Received	16%	8%	2% to 33%	--
Ignored	3.3	1.2	1 to 5	--
Excluded	3.3	1.2	1 to 5	--
Bad/Good	4.4	1.1	3 to 7	--
Sad/Happy	4.3	1.1	1 to 7	--
Unfriendly/Friendly	4.4	1.2	2 to 7	--
Relaxed/Tense	4.5	1.8	1 to 7	--
Pre-task: PANAS – Positive Affect	29.3	6.4	20 to 46	.86
Post-task: PANAS – Positive Affect	18.1	6.5	10 to 35	.87
Pre-task: PANAS – Negative Affect	14.0	3.3	10 to 25	.74
Post-task: PANAS – Negative Affect	11.6	1.8	10 to 17	.37[2]

Note. Subscales and total score for the Need-Threat Scale (NTS) are computed as the average across items. Percentage of Throws Received refers to the participants' perceptions of the percentage of throws they received during the exclusion phase of the paradigm. Cronbach's α represents observed score reliability for the present sample. The Relaxed/Tense item was reverse scored. PANAS = Positive and Negative Affect Schedule [1]Represents the average scores across items for each NTS subscale [2]Post-task PANAS – Negative Affect had low score variability and most scores were impacted by a flooring effect (note mean, standard deviation and range of scores), which resulted in low Cronbach's α.

Table 4

Scalp-Level Event-Related Potential Amplitude (µV) and Source-Level Amplitude (nAm) Summary Data as a Function of Event Type

	Inclusion Phase				Exclusion Phase			
	Inclusionary Throw		Exclusionary Throw		Exclusionary Throw – 1^{st} Block		Exclusionary Throw – 2^{nd} Block	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
N2	3.2	2.8	4.3	3.1	3.4	3.0	2.8	2.0
Frontal P3	4.0	2.9	4.6	2.8	4.1	3.6	2.4	2.0
Parietal P3	4.4	2.6	4.4	2.2	4.1	2.7	3.2	1.6
Exclusion SP	-1.9	2.2	0.3	1.7	0.7	2.1	-.4	1.3
dACC - Early	4.0	8.7	10.0	11.4	12.6	11.8	4.8	12.1
dACC - Late	28.4	14.3	36.0	24.8	34.6	14.6	28.4	16.8
Left AI - Early	6.4	13.6	-1.4	12.9	2.6	16.3	-2.4	14.0
Left AI - Late	27.1	15.5	20.0	15.3	35.3	21.1	20.8	10.9
Right AI - Early	14.6	19.0	5.0	18.0	3.2	23.4	5.1	19.8
Right AI - Late	37.0	23.0	18.2	12.9	34.6	18.1	26.0	18.0
Right vlPFC	21.7	12.6	14.8	8.8	25.6	22.7	18.2	18.7
PCC	16.2	10.0	22.4	7.9	22.9	11.4	16.4	6.7
SFG	17.7	6.2	19.5	10.2	21.1	8.7	14.2	7.7

Note. Early dACC, left AI, and right AI activity correspond to the time window for N2 activity. Late dACC activity corresponds to the time window for P3. Late left and right AI activity correspond to the time window for the exclusion SP. Exclusion SP = exclusion slow potential, dACC = dorsal anterior cingulate cortex, AI = anterior insula, vlPFC = ventrolateral prefrontal cortex, PCC = posterior cingulate cortex, SFG = superior frontal gyrus

Table 5

Zero-Order Correlations between Scalp-Level Activity and Source-Level Activity for Each Event Type

Component	Phase	Throw	dACC	Left AI	Right AI	Right vlPFC	PCC	SFG
N2	Inclusion	Inclusionary	.18	.12	.21	--	--	--
		Exclusionary	.39*	-.01	-.09	--	--	--
	Exclusion	Exclusionary	.35†	-.36*	-.18	--	--	--
Frontal P3	Inclusion	Inclusionary	.15	--	--	--	.57§	.21
		Exclusionary	.56§	--	--	--	.37*	.32†
	Exclusion	Exclusionary	.28	--	--	--	.34†	.22
Parietal P3	Inclusion	Inclusionary	.22	--	--	--	.53§	.39*
		Exclusionary	.50§	--	--	--	.51§	.26
	Exclusion	Exclusionary	.31†	--	--	--	.28	.43*
Exclusion SP	Inclusion	Inclusionary	.12	-.21	-.18	-.11	-.24	--
		Exclusionary	.50§	.16	-.07	-.12	.02	--
	Exclusion	Exclusionary	.20	-.20	-.26	-.17	.16	--

Note. Scalp-level component amplitude was correlated with source-level activity for each specified event type. Only regional source waveform temporal windows that corresponded to scalp ERP activity were examined. $^\dagger p < .10$, $^* p < .05$, $^\S p < .01$; Exclusion SP = exclusion slow potential, dACC = dorsal anterior cingulate cortex, AI = anterior insula, vlPFC = ventrolateral prefrontal cortex, PCC = posterior cingulate cortex, SFG = superior frontal gyrus

Table 6

Scalp-Level Event-Related Potential Amplitude (µV) and Source-Level Amplitude (nAm) Summary Data as a Function of Event Type for Data During the Entire Exclusion Phase

	Inclusion Phase				Exclusion Phase	
	Inclusionary Throw		Exclusionary Throw		Exclusionary Throw	
	M	*SD*	*M*	*SD*	*M*	*SD*
N2	3.1	2.7	4.2	3.0	3.0	2.2
Frontal P3	4.0	2.8	4.6	2.8	2.7	2.3
Parietal P3	4.5	2.5	4.5	2.2	3.3	1.9
Exclusion SP	-1.7	2.2	0.3	1.6	0.1	2.2
dACC - Early	4.1	8.4	10.7	11.3	3.5	11.8
dACC - Late	28.0	13.6	36.8	23.8	27.0	15.1
Left AI - Early	6.0	13.4	-2.3	15.1	-2.7	12.2
Left AI - Late	27.8	15.2	20.0	15.1	20.3	11.0
Right AI - Early	13.4	19.1	5.6	17.4	3.4	16.2
Right AI - Late	34.6	23.1	19.1	12.6	23.6	16.3
Right vlPFC	22.0	12.2	15.4	8.5	15.6	15.4
PCC	25.8	10.0	23.5	8.8	18.1	5.9
SFG	17.4	6.1	19.1	9.7	13.8	7.0

Note. Early dACC, left AI, and right AI activity correspond to the time window for N2 activity. Late dACC activity corresponds to the time window for P3. Late left and right AI activity correspond to the time window for the exclusion SP. Exclusion SP = exclusion slow potential, dACC = dorsal anterior cingulate cortex, AI = anterior insula, vlPFC = ventrolateral prefrontal cortex, PCC = posterior cingulate cortex, SFG = superior frontal gyrus

Table 7

Mean Summary Data for Self-Report Measures

	M	*SD*	Range	Cronbach's α
GTS – NTS	10.3	6.4	1 to 26	.84
GTS – PTS	16.7	6.7	1 to 27	.87
PSWQ	46.4	4.66	39 to 58	.69
MASQ – AA	21.6	4.1	17 to 34	.64
MASQ – AD	57.7	12.6	31 to 82	.91
SSQ – Average Number of People	5.0	2.2	1.3 to 8	.91
SSQ – Average Satisfaction	5.2	1.1	1.5 to 6	.93

Note. GTS – NTS = General Temperament Survey – Negative Temperament Scale, GTS – PTS = General Temperament Survey – Positive Temperament Scale, PSWQ = Penn State Worry Questionnaire, MASQ – AA = Mood and Anxiety Symptom Questionnaire – Anxious Arousal, MASQ – AD = Mood and Anxiety Symptom Questionnaire – Anhedonic Depression, SSQ = Social Support Questionnaire

Figure Captions

Figure 1. Layout of the 96-channel EEG sensors.

Figure 2. Source model showing the spatial locations of the nine regional sources included in the model. Dark grey diamonds show location of the noise sources. dACC = dorsal anterior cingulate cortex, LAI = left anterior insula, RAI = right anterior insula, vlPFC = ventrolateral prefrontal cortex, PCC = posterior cingulate cortex, SFG = superior frontal gyrus

Figure 3. Grand-average scalp waveforms averaged across A) four electrode sides for N2 activity and C) six electrode sites for exclusion slow potential (exclusion SP) activity. Voltage maps showing the average activity from B) 275 to 325 ms and D) 600 to 900 ms for difference activity for an exclusionary throw during the first block of the exclusion phase minus an exclusionary throw during the second block of the exclusion phase.

Figure 4. Grand-average scalp waveforms averaged across A) three electrode sides for frontal P3 activity and C) five electrode sites for parietal P3 activity. Voltage maps showing the average activity from B) 300 and 500 ms and D) 250 and 450 ms for difference activity for an exclusionary throw during the first block of the exclusion phase minus an exclusionary throw during the second block of the exclusion phase.

Figure 5. Grand-average source waveforms for activity during the first and second block of the exclusion phase corresponding to A) dorsal anterior cingulate cortex, B) left anterior insula, C) right anterior insula, D) right ventrolateral prefrontal cortex, E) posterior cingulate cortex, and F) superior frontal gyrus.

Figure 6. Grand-average scalp waveforms averaged across A) four electrode sides for N2 activity and C) six electrode sites for exclusion slow potential (exclusion SP) activity. Voltage maps showing the average activity from B) 275 to 325 ms and D) 600 to 900 ms for two conditions.

The left voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an inclusionary throw during the inclusion phase. The right voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an exclusionary throw during the first block of the exclusion phase.

Figure 7. Grand-average scalp waveforms averaged across A) three electrode sides for frontal P3 activity and C) five electrode sites for parietal P3 activity. Voltage maps showing the average activity from B) 300 and 500 ms and D) 250 and 450 ms for two conditions. The left voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an inclusionary throw during the inclusion phase. The right voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an exclusionary throw during the first block of the exclusion phase.

Figure 8. Grand-average source waveforms for activity corresponding to A) dorsal anterior cingulate cortex, B) left anterior insula, C) right anterior insula, D) right ventrolateral prefrontal cortex, E) posterior cingulate cortex, and F) superior frontal gyrus.

Figure 9. Grand-average scalp waveforms averaged across A) four electrode sides for N2 activity and C) six electrode sites for exclusion slow potential (exclusion SP) activity. Voltage maps showing the average activity from B) 275 to 325 ms and D) 600 to 900 ms for two conditions. The left voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an inclusionary throw during the inclusion phase. The right voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an exclusionary throw during the exclusion phase.

Figure 10. Grand-average scalp waveforms averaged across A) three electrode sides for frontal P3 activity and C) five electrode sites for parietal P3 activity. Voltage maps showing the average

activity from B) 300 and 500 ms and D) 250 and 450 ms for two conditions. The left voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an inclusionary throw during the inclusion phase. The right voltage maps show the difference activity for an exclusionary throw during the inclusion phase minus an exclusionary throw during the exclusion phase.

Figure 11. Grand-average source waveforms for activity corresponding to A) dorsal anterior cingulate cortex, B) left anterior insula, C) right anterior insula, D) right ventrolateral prefrontal cortex, E) posterior cingulate cortex, and F) superior frontal gyrus.

Figure 1

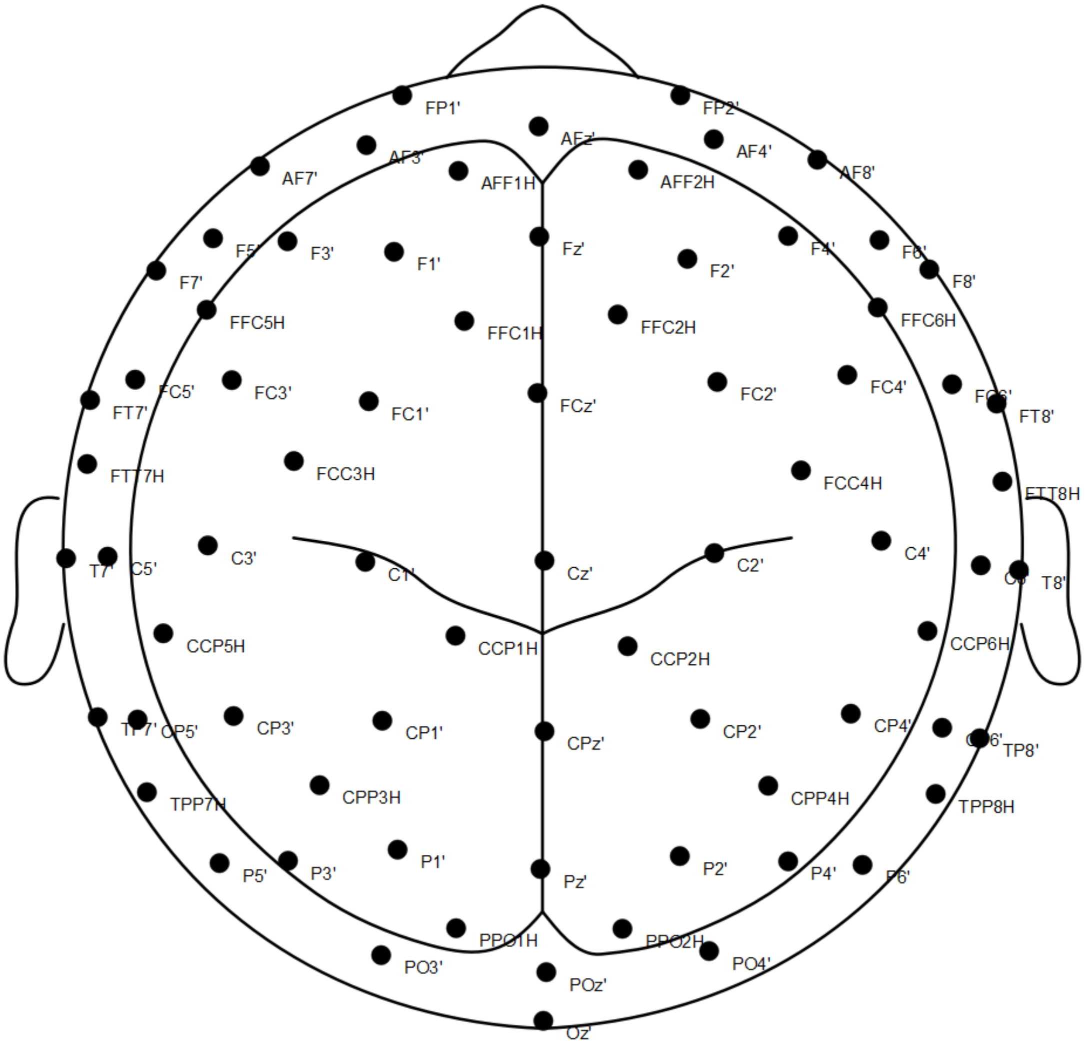
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Figure 2

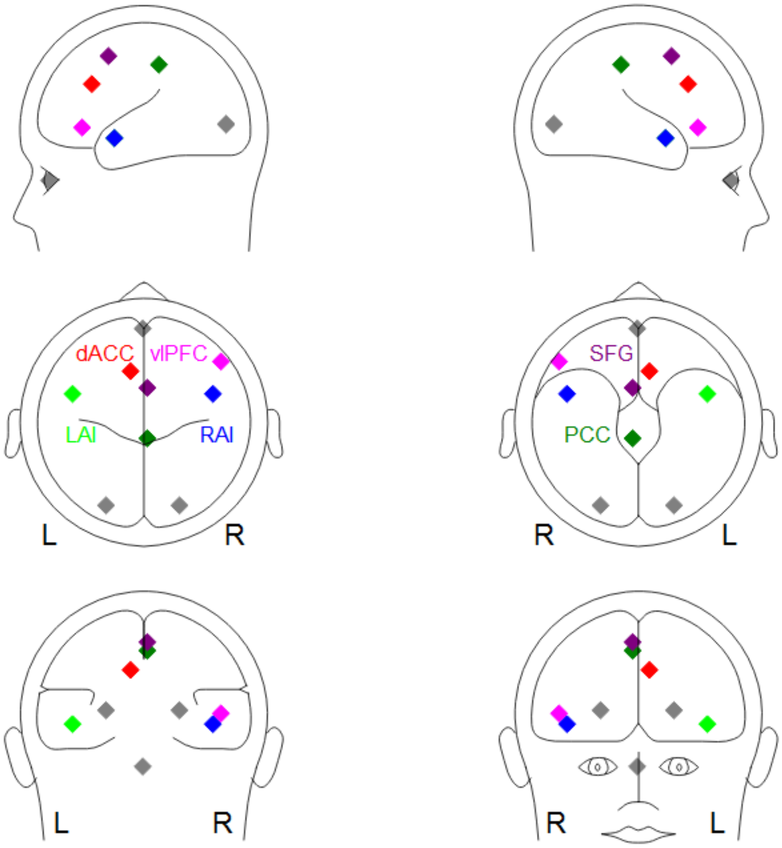
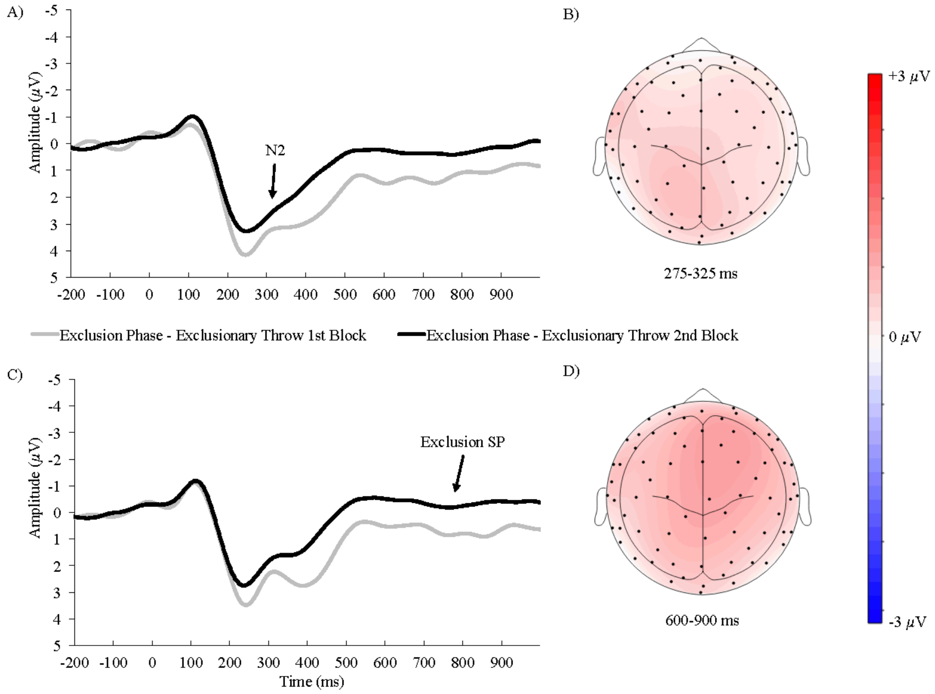


Figure 3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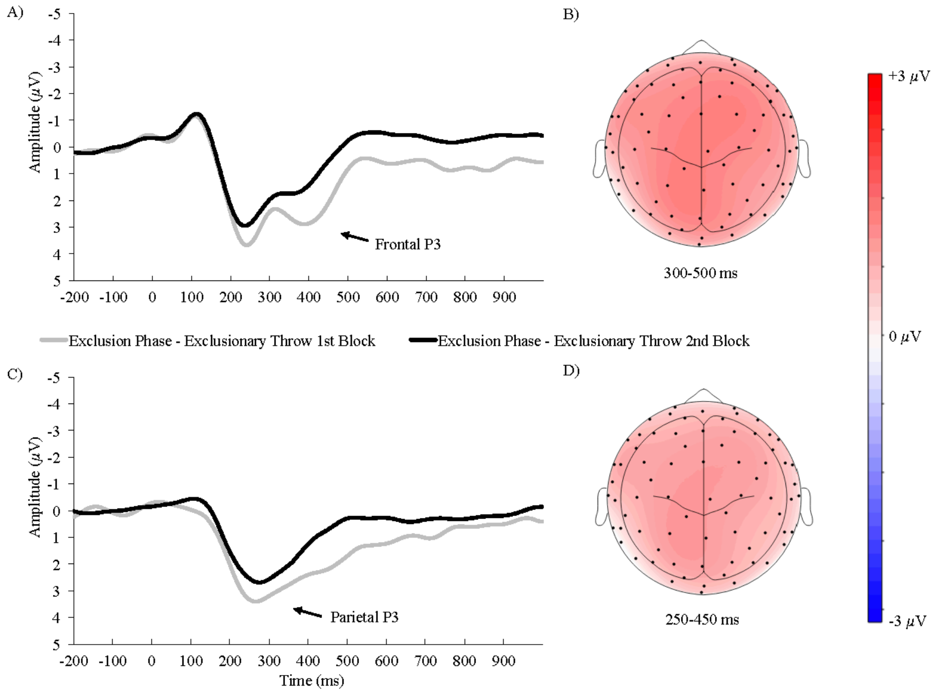
100
200
300
400
500
600
700
800
900
N2
B)
275-325 ms
+3 μV
0 μV
-3 μV
Exclusion Phase - Exclusionary Throw 1st Block
Exclusion Phase - Exclusionary Throw 2nd Block
C)
Amplitude (μV)
Exclusion SP
Time (ms)
D)
600-900 ms

Figure 4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B)
C)
D)
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300
400
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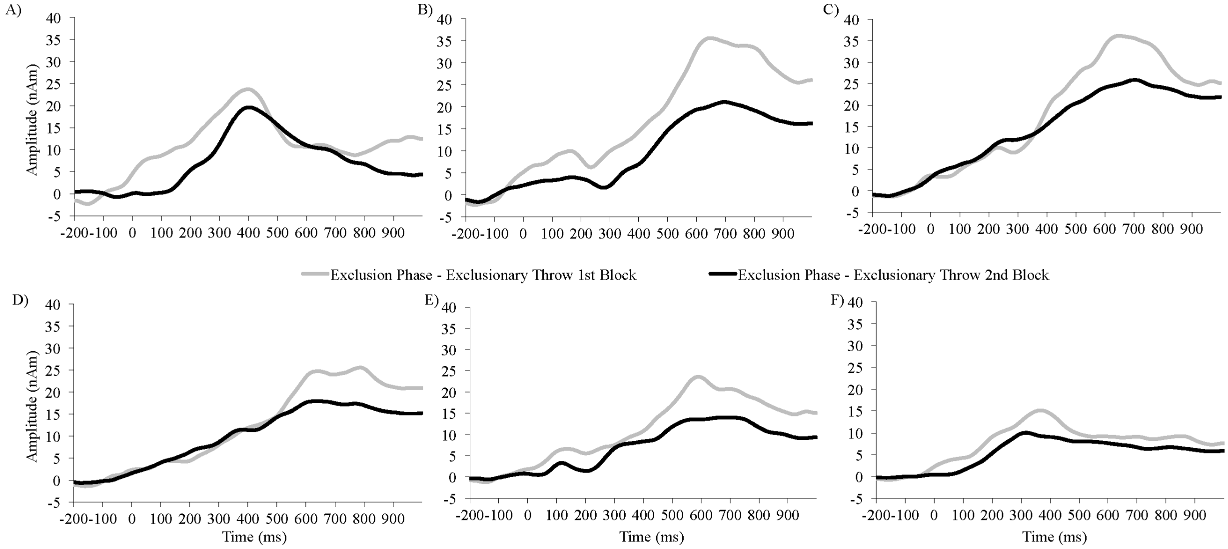
600
700
800
900
Frontal P3
Parietal P3
Time (ms)
300-500 ms
250-450 ms
+3 µV
0 µV
-3 µV
Exclusion Phase - Exclusionary Throw 1st Block
Exclusion Phase - Exclusionary Throw 2nd Block

Figure 5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D)
E)
F)
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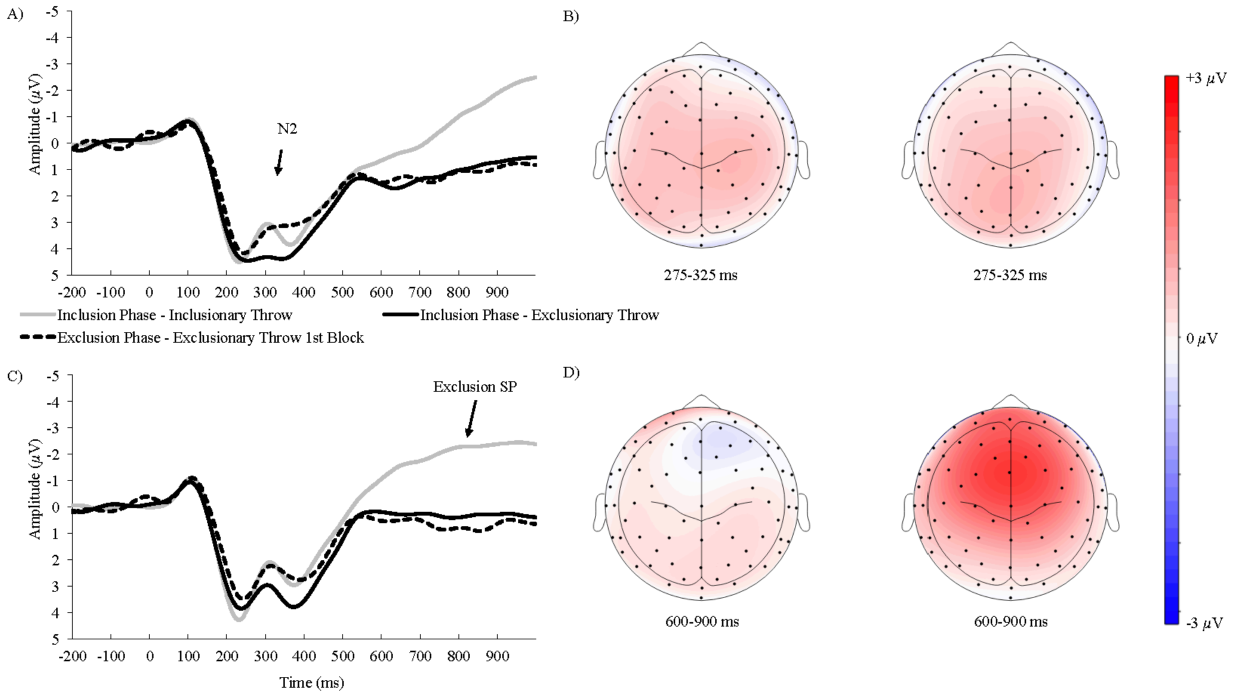
Time (ms)
Exclusion Phase - Exclusionary Throw 1st Block
Exclusion Phase - Exclusionary Throw 2nd Block

Figure 6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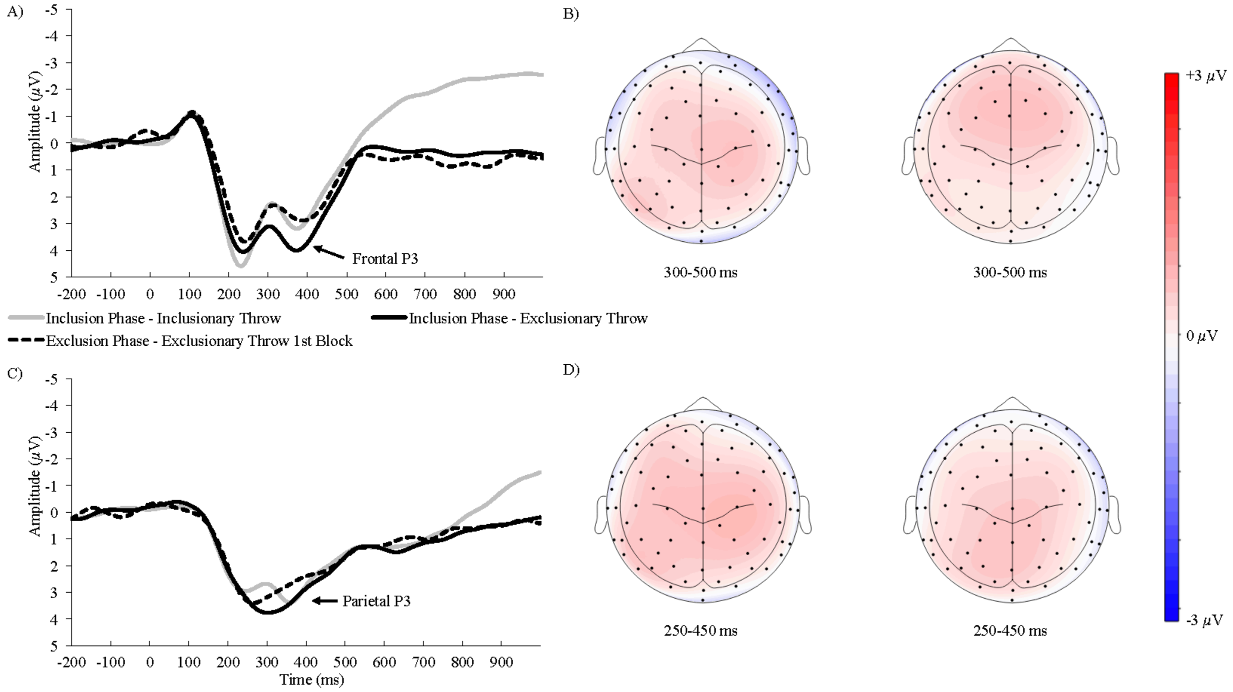
N2
Inclusion Phase - Inclusionary Throw
Inclusion Phase - Exclusionary Throw
Exclusion Phase - Exclusionary Throw 1st Block
B)
275-325 ms
275-325 ms
C)
Exclusion SP
Time (ms)
D)
600-900 ms
600-900 ms
+3 μV
0 μV
-3 μV

Figure 7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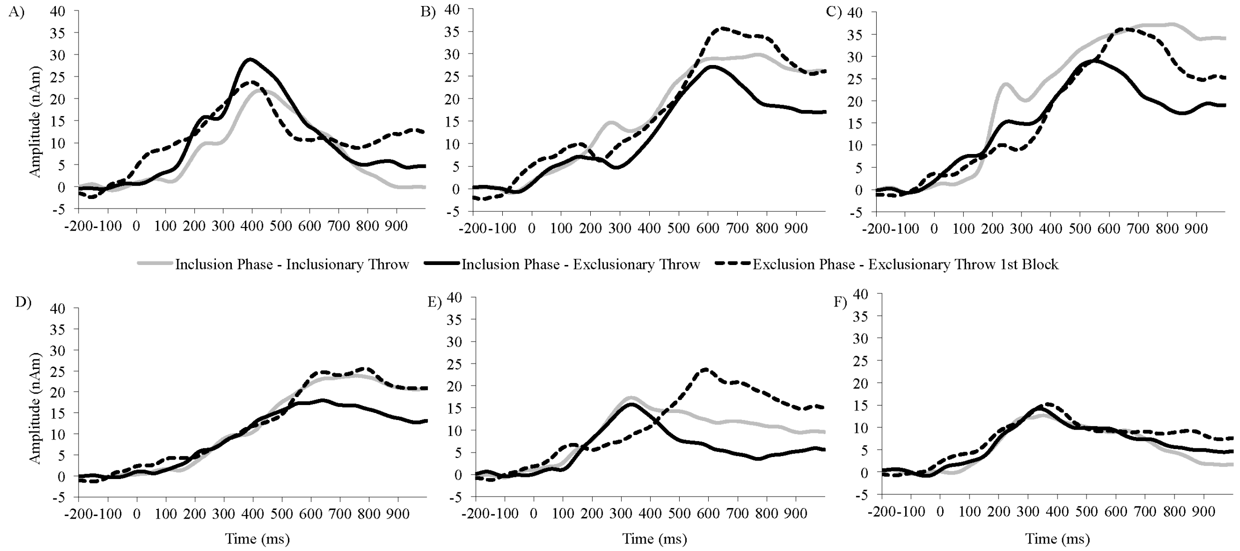
600
700
800
900
Frontal P3
Inclusion Phase - Inclusionary Throw
Inclusion Phase - Exclusionary Throw
Exclusion Phase - Exclusionary Throw 1st Block
B)
300-500 ms
300-500 ms
+3 μV
0 μV
-3 μV
C)
Amplitude (μV)
Parietal P3
Time (ms)
D)
250-450 ms
250-450 ms

Figure 8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C)
D)
E)
F)
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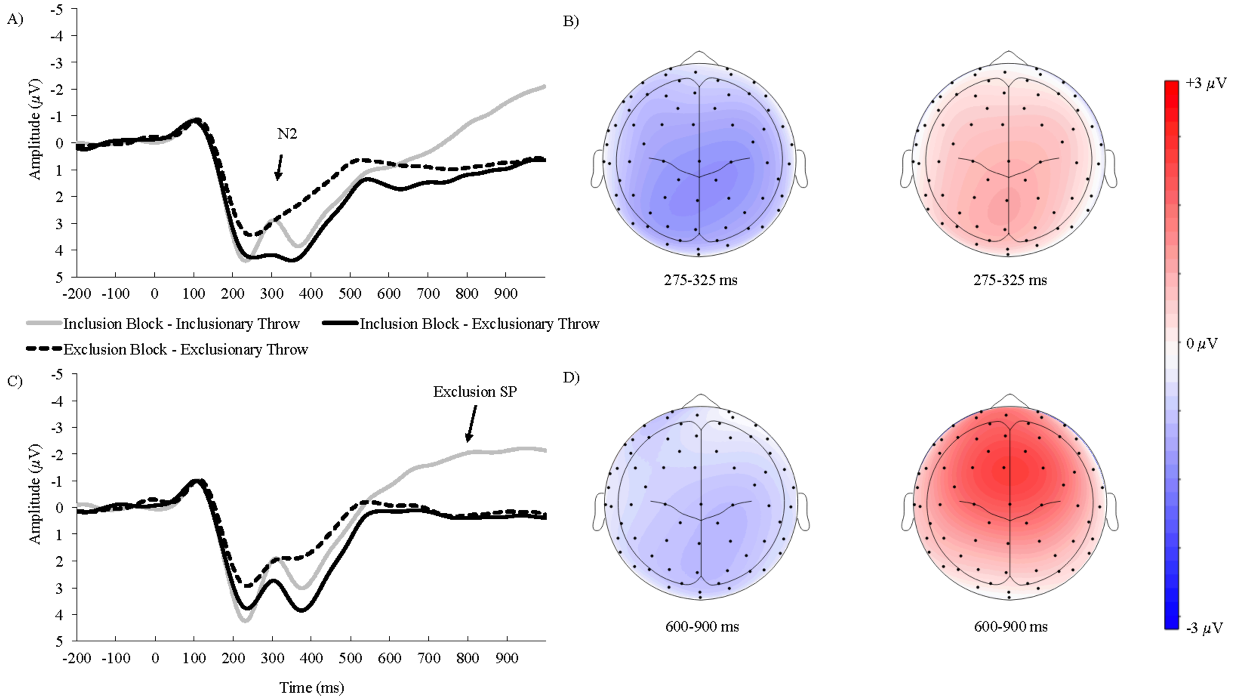
-5
-200-100 0 100 200 300 400 500 600 700 800 900
Time (ms)
Inclusion Phase - Inclusionary Throw
Inclusion Phase - Exclusionary Throw
Exclusion Phase - Exclusionary Throw 1st Block

Figure 9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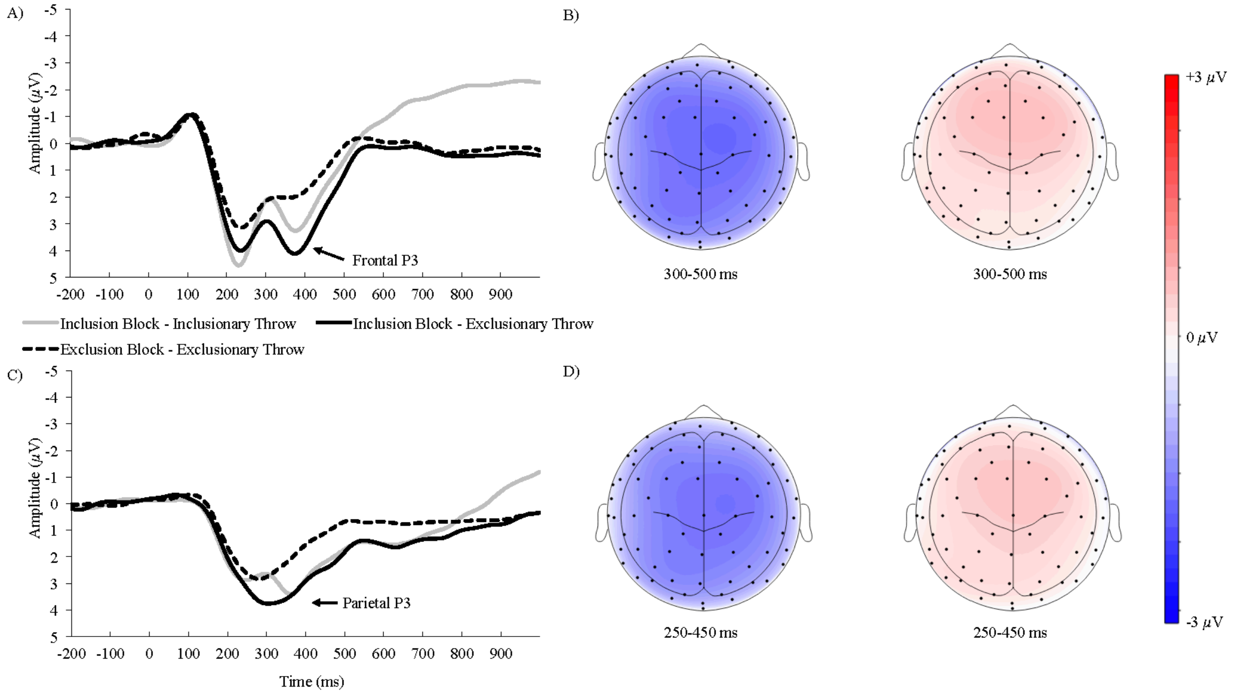
700
800
900
N2
Exclusion SP
Time (ms)
Inclusion Block - Inclusionary Throw
Inclusion Block - Exclusionary Throw
Exclusion Block - Exclusionary Throw
275-325 ms
275-325 ms
600-900 ms
600-900 ms
+3 μV
0 μV
-3 μV

Figure 10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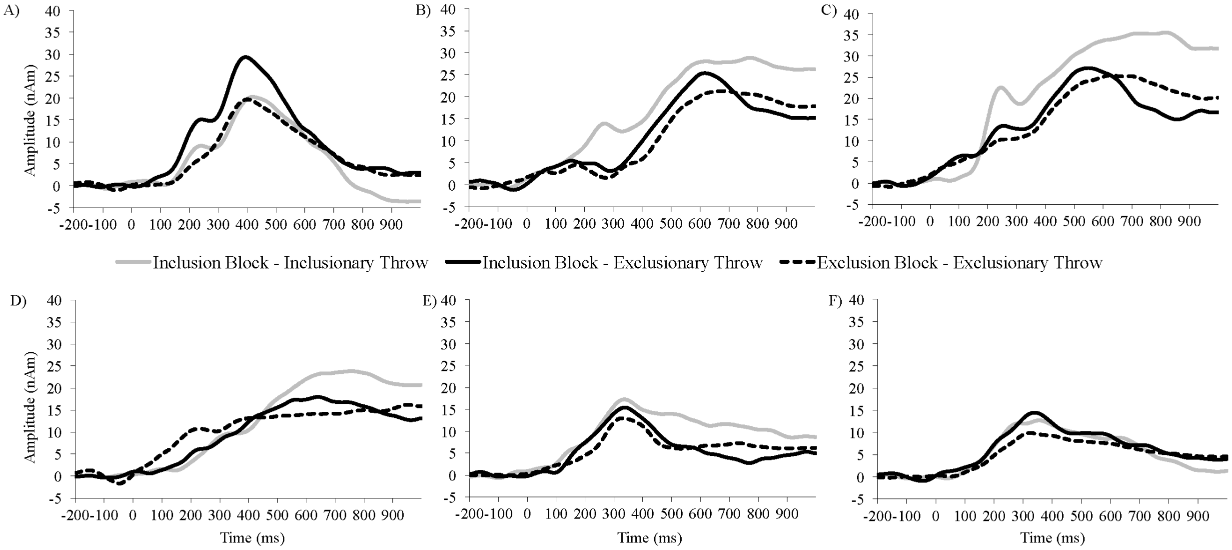
600
700
800
900
Frontal P3
Parietal P3
Inclusion Block - Inclusionary Throw
Inclusion Block - Exclusionary Throw
Exclusion Block - Exclusionary Throw
Time (ms)
300-500 ms
300-500 ms
250-450 ms
250-450 ms
+3 μV
0 μV
-3 μV

Figure 11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B)
C)
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F)
Amplitude (nAm)
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35
30
25
20
15
10
5
0
-5
-200-100 0 100 200 300 400 500 600 700 800 900
Time (ms)
Inclusion Block - Inclusionary Throw
Inclusion Block - Exclusionary Throw
Exclusion Block - Exclusionary Throw

Appendix

Analyses below include the data for all exclusionary throws during the exclusion phase of the Cyberball task.

ERP Component Analyses

Grand-average scalp activity waveforms and topographic maps are presented in Figures 9 and 10. Summary data for scalp-activity component amplitudes are presented in Table 6.

N2. Contrary to the predicted pattern in hypothesis 1a, a robust ANOVA on N2 amplitudes for inclusionary and exclusionary throws during the inclusion phase indicated that N2 amplitude was more negative for inclusionary throws than for exclusionary throws, $T_{WJt}/c(1.0,31.0)=10.71$, $p=.002$. Also contrary to hypothesis 1a and to the pattern observed for N2 scores from the first block of the exclusion phase, N2 amplitude was more negative for exclusionary throws during the exclusion phase than for exclusionary throws during the inclusion phase, $T_{WJt}/c(1.0,31.0)=7.71$, $p=.007$. Such a pattern is suggestive of N2 being at least partially sensitive to social exclusion.

Frontal P3. A significant difference between inclusionary and exclusionary throws during the inclusion phase was not observed for frontal P3 amplitudes, as indicated by the nonsignificant robust ANOVA, $T_{WJt}/c(1.0,31.0)=2.00$, $p=.17$. Contrary to hypothesis 2b and to the findings from the analyses using exclusionary throws during the first block of the exclusion phase, frontal P3 amplitude was more positive for exclusionary throws during the inclusion phase than for exclusionary throws during the exclusion phase, $T_{WJt}/c(1.0,31.0)=30.6$, $p<.0001$.

Parietal P3. The robust ANOVA on parietal P3 amplitudes during the inclusion phase yielded a nonsignificant effect, indicating that similar amplitudes were observed for inclusionary and exclusionary throws, $T_{WJt}/c(1.0,31.0)=0.01$, $p=.94$. Contrary to hypothesis 2b and inconsistent with findings from the analyses of exclusionary throws during the first block of the exclusion phase, parietal P3 amplitude was more positive for exclusionary throws during the inclusion phase than for exclusionary throws during the exclusion phase, $T_{WJt}/c(1.0,31.0)=10.23$, $p=.002$.

Exclusion SP. A robust ANOVA on exclusion SP amplitudes for inclusionary and exclusionary throws during the inclusion phase indicated that exclusion SP amplitude was more negative for inclusionary throws than for exclusionary throws, $T_{WJt}/c(1.0,31.0)=20.07$, $p<.001$. Contrary to hypothesis 2a, exclusion SP amplitudes were similar for exclusionary throws during the inclusion and exclusion phases, $T_{WJt}/c(1.0,31.0)=0.11$, $p=.75$. This finding was also consistent with the analyses only using the first block of exclusionary throws from the exclusion phase.

Source Waveform Analyses

Grand-average source waveforms are presented in Figure 11. Summary data for source component amplitudes are presented in Table 6.

dACC. The first scoring window for dACC corresponded to scalp-recorded N2 activity. A robust ANOVA comparing source-waveform peaks during inclusionary and exclusionary throws during the inclusion phase indicated that dACC activity was larger for exclusionary throws than for inclusionary throws, $T_{WJt}/c(1.0,30.0)=8.52$, $p=.006$. Inconsistent with findings from the data using only the first block of the exclusion phase, dACC activity for exclusionary throws during the inclusion phase was larger than during the exclusion phase, $T_{WJt}/c(1.0,30.0)=8.05$, $p=.006$. dACC differentiated between inclusionary and exclusionary throws in the inclusion phase and between exclusionary throws during the inclusion and exclusion phases.

The second scoring window for dACC corresponded to frontal P3 activity. Consistent with findings from the analyses examining the first block of the exclusion phase, robust ANOVA indicated that exclusionary throws during the inclusion phase were larger than inclusionary throws during the inclusion phase, $T_{\mathrm{WJt}}/c(1.0,30.0)=6.27$, $p=.02$. Contrary to the nonsignificant findings observed for the analyses examining the first block of the exclusion phase, dACC activity for exclusionary throws during the inclusion phase were also larger than the exclusionary throws during the exclusion phase, $T_{\mathrm{WJt}}/c(1.0,30.0)=8.65$, $p=.01$. However, this pattern of effects was the opposite of the pattern predicted in hypothesis 2d, which predicted greater activity during the exclusion phase than during the inclusion phase.

Left AI. The first scoring window for the left AI source corresponded to N2 activity. Inconsistent with the analyses that examined the first block of the exclusion phase, a robust ANOVA for left AI activity during the inclusion phase indicated greater activity for inclusionary throws than for exclusionary throws, $T_{\mathrm{WJt}}/c(1.0,30.0)=5.84$, $p=.03$. Consistent with analyses of the first block of the exclusion phase, left AI activity was similar for exclusionary throws during the inclusion and exclusion phases, $T_{\mathrm{WJt}}/c(1.0,30.0)=0.01$, $p=.90$.

The second scoring window for left AI corresponded to exclusion SP activity. Paralleling findings using the first block of the exclusion phase, left AI activity was greater for inclusionary throws during the inclusion phase than for exclusionary throws during the inclusion phase, $T_{\mathrm{WJt}}/c(1.0,30.0)=5.77$, $p=.03$. Contrary to the analyses using the first block of throws from the exclusion phase, the exclusionary throws during the inclusion phase showed similar amplitudes to exclusionary throws during the exclusion phase, $T_{\mathrm{WJt}}/c(1.0,30.0)=0.01$, $p=.91$.

Right AI. The first scoring window for right AI corresponded to N2 activity. Both analyses for early right AI activity were consistent with the analyses using data from the first block of the exclusion phase. The robust ANOVA for right AI activity for inclusionary and exclusionary throws during the inclusion phase was not significant, $T_{WJt}/c(1.0,30.0)=2.96$, $p=.11$. Right AI activity was also inconsistent with scalp N2 effects across blocks and was not significant when comparing exclusionary throws during the inclusion phase to exclusionary throws during the exclusion phase, $T_{WJt}/c(1.0,30.0)=0.30$, $p=.60$.

The second scoring window for right AI corresponded to exclusion SP activity and showed a pattern similar to exclusion SP effects. Similar to the analyses using the first block of the exclusion phase, right AI activity was greater for inclusionary throws during the inclusion phase than for exclusionary throws during the inclusion phase, $T_{WJt}/c(1.0,30.0)=12.93$, $p=.003$. Contrary to the analyses using data from the first block of the exclusion phase, similar amplitudes were observed for the comparison of the exclusionary throws during the inclusion phase to exclusionary throws during the exclusion phase, $T_{WJt}/c(1.0,30.0)=1.92$, $p=.18$.

Right vlPFC. The scoring window for right vlPFC corresponded to exclusion SP activity. Paralleling findings using the first block of the exclusion phase, greater activation was observed during inclusionary throws than during exclusionary throws, as indicated by a robust ANOVA on right vlPFC activity for throws during the inclusion phase, $T_{WJt}/c(1.0,30.0)=9.88$, $p=.008$. Unlike the analyses of exclusionary throws from the first block of the exclusion phase, significant differences were not observed for exclusionary throws during the inclusion and exclusion phases, $T_{WJt}/c(1.0,30.0)=0.03$, $p=.87$.

PCC. The scoring window for PCC corresponded to parietal P3 activity. Consistent with analyses of the first block of the exclusion phase, a robust ANOVA for PCC activity during the inclusion phase indicated a nonsignificant difference between inclusionary and exclusionary throws, $T_{WJt}/c(1.0,30.0)=2.14$, $p=.15$. Unlike the analyses of the data using the first block of the exclusion phase, the robust ANOVA for PCC activity for exclusionary throws yielded a significant effect, with larger activity for exclusionary throws during the inclusion phase than for exclusionary throws during the exclusion phase, $T_{\mathrm{WJt}}/c(1.0,30.0)=10.78$, $p=.009$.

SFG. The scoring window for SFG also corresponded to parietal P3 activity. Similar to the analyses using the exclusionary throws during the first block of the exclusion phase, a robust ANOVA on SFG activity for throws during the inclusion phase yielded a nonsignificant effect, $T_{\mathrm{WJt}}/c(1.0,30.0)=2.00$, $p=.18$. When comparing exclusionary throws across phases, SFG activity was larger to exclusionary throws during the inclusion phase than to exclusionary throws during the exclusion phase, $T_{\mathrm{WJt}}/c(1.0,30.0)=16.45$, $p=.004$. This latter comparison was inconsistent with analyses using the data from the first block of the exclusion phase.

Correlational Analyses

Scalp- and source-level activity for exclusionary throws during the exclusion phase were compared to self-reported social distress, changes in PANAS state negative and positive affect scores, and mood item scores. For the NTS, higher NTS Total scores were associated with higher late right AI activity and right vlPFC activity, $r(29)=.39$, $p=.02$; $r(29)=.47$, $p=.007$, respectively. A temporal dissociation effect for right AI activity and a lateralization effect for late AI activity and NTS Total scores were not observed ($|Z\text{s}| < 0.50$, $p\text{s} > .61$). None of the remaining correlations with NTS Total scores was significant ($|r\text{s}| < .34$, $p\text{s} > .05$). Consistent with hypothesis 1b and analyses of exclusionary throws during the first block of the exclusion phase, N2 and early dACC activity were not significantly related to any self-report measures of social distress. Contrary to hypothesis 2c, exclusion SP and frontal P3 amplitudes were not significantly correlated with social distress scores, and these findings are similar to the analyses of exclusionary throws during the exclusion phase.

In order to examine the dimensional relationship between changes in PANAS scores and sensor- and source-space amplitude scores, PANAS difference scores (post-task minus pre-task) were calculated separately for state negative and positive affect. A greater decrease in PANAS state negative affect scores after the task was related to greater early and late right AI activity, *r*(29)=-.42, *p*=.02; *r*(29)=-.48, *p*=.007, respectively. Significant lateralization effects were not observed for early or late AI activity, and early and late right AI activity showed similar correlations to PANAS state negative affect scores (|*Z*s| < 0.53, *p*s > .59). A greater decrease in PANAS state positive affect scores after the task was related to larger parietal P3 amplitude and early left AI activity, *r*(30)=-.46, *p*=.008; *r*(29)=-.58, *p*<.001, respectively. Early AI activity showed a marginally significant lateralization effect, with a somewhat larger relationship between PANAS state positive affect scores and early left AI activity than between PANAS state positive affect scores and early right AI activity, *Z*=-1.9, *p*=.06. The remaining measures were not significantly correlated with PANAS state negative or positive affect difference scores (|*r*s| < .36, *p*s > .05).

For the Sad-Happy mood score, higher late dACC activity and SFG activity were related to lower scores, which were consistent with a sad mood score, *r*(29)=-.46, *p*=.01; *r*(29)=-.43, *p*=.02, respectively. The relationship between late dACC activity and Sad-Happy mood scores was stronger than the relationship between early dACC activity and Sad-Happy mood scores, *Z*=-2.74, *p*=.006. Higher late dACC activity was also related to lower Unfriendly-Friendly mood scores, which were consistent with an unfriendly mood score, *r*(29)=-.41, *p*=.02. The relationship between late dACC activity and Unfriendly-Friendly mood scores was stronger than the relationship between early dACC activity and Unfriendly-Friendly mood scores, *Z*=-2.47, *p*=.01. Higher early left AI activity was related to lower Relaxed-Tense mood scores, which were consistent with a relaxed mood score, *r*(29)=-.39, *p*=.03. Lateralization effects were not observed for early AI activity, *Z*=-1.0, *p*=.32. None of the remaining ERP component or source waveform scores was significantly correlated with any mood scores (|*r*s| < .35, *p*s > .05). Notably, late dACC activity showed a stronger relationship to Sad-Happy and Unfriendly-Friendly mood scores than did early dACC activity. This pattern of findings provides some evidence that late, rather early, dACC activity is related to changes in self-reported affective state.

The extent to which social support, as measured by the SSQ, related to scalp- and source-space scores was also examined. Higher levels of social support as measured by the SSQ were related to decreased right SFG activity for exclusionary throws during the exclusion phase, *r*(29)=-.38, *p*=.03. Higher ratings of satisfaction of social support were related to higher early left AI activity, *r*(29)=.36, *p*=.045. Lateralization effects for AI and temporal dissociation effects for left AI activity were not observed (|*Z*s| < 1.2, *p*s > .21). The remaining correlations were not significant (|*r*s| < .34, *p*s > .06). Contrary to hypothesis 2e, exclusion SP, frontal P3, and dACC activity were unrelated to levels of social support.

Exploratory Analyses

Summary data for GTS, PSWQ, and MASQ are presented in Table 7. Scalp- and source-space amplitude scores were compared to trait levels of positive and negative affect, as assessed by the GTS-PTS and GTS-NTS, respectively. Although the correlations mentioned below were significant at α = .05 level, they failed to meet a Bonferroni-corrected threshold. These correlations should be considered exploratory and require future replication efforts to confirm the observed relationships. Higher GTS-PTS scores were related to lower late dACC activity for exclusionary throws during the inclusion and exclusion phases, *r*(29)=-.38, *p*=.04; *r*(29)=-.45, *p*=.01, respectively. A temporal dissociation between early and late dACC activity was not observed for exclusionary throws during the inclusion and exclusion phase, *Z*=-0.50, *p*=.62; *Z*=-1.77, *p*=.08, respectively. Higher GTS-PTS scores were also related to lower right vlPFC activity for exclusionary throws during the inclusion phase and lower SFG activity for exclusionary throws during the exclusion phase, *r*(29)=-.41, *p*=.02; *r*(29)=-.46, *p*=.009, respectively. The remaining correlations with GTS-PTS scores and GTS-NTS scores were not significant (|*r*s| < .34, *p*s > .06).

Anxiety and depression scores were also correlated with scalp- and source-space amplitude scores using the PSWQ, MASQ-AA, and MASQ-AD scales. Higher PSWQ scores were related to higher late dACC activity and early right AI activity for exclusionary throws during the exclusion phase, *r*(29)=.40, *p*=.02; *r*(29)=.41, *p*=.02, respectively. Higher PSWQ scores were also related to higher right vlPFC activity for inclusionary throws during the inclusion phase, *r*(29)=.40, *p*=.02, and higher late right AI activity for exclusionary throws during the exclusion phase, *r*(29)=.37, *p*=.04. Temporal dissociations between early and late dACC and AI activity and lateralization effects for AI were not observed (|*Z*s| < 1.7, *p*s > .09). None of the remaining correlations was significant with PSWQ scores (|*r*s| < .34, *p*s > .06). Higher MASQ-AA scores were related to lower N2 and frontal P3 amplitudes for inclusionary throws during the inclusion phase, *r*(30)=-.35, *p*=.049; *r*(30)=-.36, *p*=.04, respectively. Higher MASQ-AA scores were also related to lower PCC activity for inclusion throws during the inclusion phase, *r*(29)=-.39, *p*=.03. None of the remaining correlations with MASQ-AA was significant (|*r*s| < .28, *p*s > .12). Higher MASQ-AD scores were related to higher right vlPFC activity for inclusion throws during the inclusion phase, *r*(30)=.44, *p*=.01. None of the remaining correlations with MASQ-AD was significant (|*r*s| < .32, *p*s > .07).

References

Aguirre, G. K., Zarahn, E., & D'Esposito, M. (1998). The variability of human, BOLD hemodynamic responses. *NeuroImage, 8*, 360-369. doi:10.1006/nimg.1998.0369

Akalin Acar, Z., & Makeig, S. (2013). Effects of forward model errors on EEG source localization. *Brain Topography, 26*, 378-396. doi:10.1007/s10548-012-0274-6

Alonso, J., Buron, A., Rojas-Farreras, S., de Graaf, R., Haro, J. M., de Girolamo, G., . . . Vilagut, G. (2009). Perceived stigma among individuals with common mental disorders. *Journal of Affective Disorders, 118*, 180-186. doi:10.1016/j.jad.2009.02.006

Apkarian, A. V., Bushnell, M. C., Treede, R.-D., & Zubieta, J.-K. (2005). Human brain mechanisms of pain perception and regulation in health and disease. *European Journal of Pain, 9*, 463-484. doi:10.1016/j.ejpain.2004.11.001

Baker, B., Kazarian, S. S., Helmes, E., Ruckman, M., & Tower, N. (1987). Perceived attitudes of schizophrenic inpatients in relation to rehospitalization. *Journal of Consulting and Clinical Psychology, 55*, 775-777. doi:10.1037/0022-006X.55.5.775

Baker, D. G., Nievergelt, C. M., & O'Connor, D. T. (2012). Biomarkers of PTSD: Neuropeptides and immune signaling. *Neuropharmacology, 62*, 663-673. doi:10.1016/j.neuropharm.2011.02.027

Bandettini, P. A., Wong, E. C., Hinks, R. S., Tikofsky, R. S., & Hyde, J. S. (1992). Time course EPI of human brain function during task activation. *Magnetic Resonance in Medicine, 25*, 390-397. doi:10.1002/mrm.1910250220

Banich, M. T. (2009). Executive function: The search for an integrated account. *Current Directions in Psychological Science, 18*, 89-94. doi:10.1111/j.1467-8721.2009.01615.x

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117*, 497-529. doi:10.1037/0033-2909.117.3.497

Beck, J. C., & Worthen, K. (1972). Precipitating stress, crisis theory, and hospitalization in schizophrenia and depression. *Archives of General Psychiatry, 26*, 123-129. doi:10.1001/archpsyc.1972.01750200027006

Beltrachini, L., von Ellenrieder, N., & Muravchik, C. H. (2011). General bounds for electrode mislocation on the EEG inverse problem. *Computer Methods and Programs in Biomedicine, 103*, 1-9. doi:10.1016/j.cmpb.2010.05.008

Bengtsson-Tops, A., & Hansson, L. (2001). Quantitative and qualitative aspects of the social network in schizophrenic patients living in the community. Relationship to sociodemographic characteristics and clinical factors and subjective quality of life. *The International Journal of Social Psychiatry, 47*, 67-77. doi:10.1177/002076400104700307

Berg, P., & Scherg, M. (1994a). A fast method for forward computation of multiple-shell spherical head models. *Electroencephalography and Clinical Neurophysiology, 90*, 58-64. doi:10.1016/0013-4694(94)90113-9

Berg, P., & Scherg, M. (1994b). A multiple source approach to the correction of eye artifacts. *Electroencephalography and Clinical Neurophysiology, 90*, 229-241. doi:10.1016/0013-4694(94)90094-9

Bergen, J., Hunt, G., Armitage, P., & Bashir, M. (1998). Six-month outcome following a relapse of schizophrenia. *The Australian and New Zealand Journal of Psychiatry, 32*, 815-822.

Berthier, M., Starkstein, S., & Leiguarda, R. (1988). Asymbolia for pain: A sensory ‐ limbic disconnection syndrome. *Annals of Neurology, 24*, 41-49. doi:10.1002/ana.410240109

Boksem, M. A. S., Meijman, T. F., & Lorist, M. M. (2006). Mental fatigue, motivation and action monitoring. *Biological Psychology, 72*, 123-132. doi:10.1016/j.biopsycho.2005.08.007

Botvinick, M. W. (2007). Conflict monitoring and decision making: Reconciling two perspectives on anterior cingulate function. *Cognitive, Affective & Behavioral Neuroscience, 7*, 356-366. doi:10.3758/CABN.7.4.356

Botvinick, M. W., Carter, C. S., Braver, T. S., Barch, D. M., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*, 624-652. doi:10.1037/0033-295X.108.3.624

Botvinick, M. W., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences, 8*, 539-546. doi:10.1016/j.tics.2004.10.003

Bourque, F., van der Ven, E., & Malla, A. (2011). A meta-analysis of the risk for psychotic disorders among first- and second-generation immigrants. *Psychological Medicine, 41*, 897-910. doi:10.1017/S0033291710001406

Boynton, G. M., Engel, S. A., Glover, G. H., & Heeger, D. J. (1996). Linear systems analysis of functional magnetic resonance imaging in human V1. *The Journal of Neuroscience, 16*, 4207-4221.

Brazdil, M., Roman, R., Daniel, P., & Rektor, I. (2005). Intracerebral error-related negativity in a simple go/no-go task. *Journal of Psychophysiology, 19*, 244-255. doi:10.1027/0269-8803.19.4.244

Bresnahan, M., Begg, M. D., Brown, A., Schaefer, C., Sohler, N., Insel, B., . . . Susser, E. (2007). Race and risk of schizophrenia in a US birth cohort: Another example of health disparity? *International Journal of Epidemiology, 36*, 751-758. doi:10.1093/ije/dym041

Brown, G. W., Birley, J. L., & Wing, J. K. (1972). Influence of family life on the course of schizophrenic disorders: A replication. *The British Journal of Psychiatry 121*, 241-258. doi:10.1192/bjp.121.3.241

Brown, J. W. (2013). Beyond conflict monitoring: Cognitive control and the neural basis of thinking before you act. *Current Directions in Psychological Science, 22*, 179-185. doi:10.1177/0963721412470685

Buchanan, J. (1995). Social support and schizophrenia: A review of the literature. *Archives of Psychiatric Nursing, 9*, 68-76. doi:10.1016/S0883-9417(95)80003-4

Burklund, L. J., Eisenberger, N. I., & Lieberman, M. D. (2007). The face of rejection: Rejection sensitivity moderates dorsal anterior cingulate activity to disapproving facial expressions. *Social Neuroscience, 2*, 238-253. doi:10.1080/17470910701391711

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*, 215-222. doi:10.1016/s1364-6613(00)01483-2

Butzlaff, R. L., & Hooley, J. M. (1998). Expressed emotion and psychiatric relapse: A meta-analysis. *Archives of General Psychiatry, 55*, 547-552. doi:10.1001/archpsyc.55.6.547

Cacioppo, J. T., & Hawkley, L. C. (2009). Perceived social isolation and cognition. *Trends in Cognitive Sciences, 13*, 447-454. doi:10.1016/j.tics.2009.06.005

Cantor-Graae, E., & Selten, J.-P. (2005). Schizophrenia and migration: A meta-analysis and review. *The American Journal of Psychiatry, 162*, 12-24. doi:10.1176/appi.ajp.162.1.12

Carpenter, B., Gelman, A., Hoffman, M., Lee, D., Goodrich, B., Betancourt, M., . . . Riddell, A. (2017). Stan: A probabilistic programming language. *Journal of Statistical Software, 76*. doi:10.18637/jss.v076.i01

Carter, C. S., & van Veen, V. (2007). Anterior cingulate cortex and conflict detection: An update of theory and data. *Cognitive Affective, and Behavioral Neuroscience, 7*, 367-379. doi:10.3758/CABN.7.4.367

Clawson, A., Clayson, P. E., & Larson, M. J. (2013). Cognitive control adjustments and conflict adaptation in major depressive disorder. *Psychophysiology, 50*, 711-721. doi:10.1111/psyp.12066

Clayson, P. E., Baldwin, S. A., & Larson, M. J. (2013). How does noise affect amplitude and latency measurement of event-related potentials (ERPs)? A methodological critique and simulation study. *Psychophysiology, 50*, 174-186. doi:10.1111/psyp.12001

Clayson, P. E., & Miller, G. A. (2017a). ERP Reliability Analysis (ERA) Toolbox: An open-source toolbox for analyzing the reliability of event-related potentials. *International Journal of Psychophysiology, 111*, 68-79. doi:10.1016/j.ijpsycho.2016.10.012

Clayson, P. E., & Miller, G. A. (2017b). Psychometric considerations in the measurement of event-related brain potentials: Guidelines for measurement and reporting. *International Journal of Psychophysiology, 111*, 57-67. doi:10.1016/j.ijpsycho.2016.09.005

Cohen, C. I., & Sokolovsky, J. (1978). Schizophrenia and social networks: Ex-patients in the inner city. *Schizophrenia Bulletin, 4*, 546-560. doi:10.1093/schbul/4.4.546

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*, 215-229. doi:10.1038/nrn755

Craig, A. D. (2002). How do you feel? Interoception: The sense of the physiological condition of the body. *Nature Reviews Neuroscience, 3*, 655-666. doi:10.1038/nrn894

Craig, A. D., Reiman, E. M., Evans, A., & Bushnell, M. C. (1996). Functional imaging of an illusion of pain. *Nature, 384*, 258-260. doi:10.1038/384258a0

Crawford, J. R., & Henry, J. D. (2004). The positive and negative affect schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *The British Journal of Clinical Psychology, 43*, 245-265. doi:10.1348/0144665031752934

Crottaz-Herbette, S., & Menon, V. (2006). Where and when the anterior cingulate cortex modulates attentional response: Combined fMRI and ERP evidence. *Journal of Cognitive Neuroscience, 18*, 766-780. doi:10.1162/jocn.2006.18.5.766

Crowley, M. J., Wu, J., McCarty, E. R., David, D. H., Bailey, C. A., & Mayes, L. C. (2009). Exclusion and micro-rejection: Event-related potential response predicts mitigated distress. *NeuroReport, 20*, 1518-1522. doi:10.1097/WNR.0b013e328330377a

Crowley, M. J., Wu, J., Molfese, P. J., & Mayes, L. C. (2010). Social exclusion in middle childhood: Rejection events, slow-wave neural activity, and ostracism distress. *Social Neuroscience, 5*, 483-495. doi:10.1080/17470919.2010.500169

Cutting, L. P., Aakre, J. M., & Docherty, N. M. (2006). Schizophrenic patients' perceptions of stress, expressed emotion, and sensitivity to criticism. *Schizophrenia Bulletin, 32*, 743-750. doi:10.1093/schbul/sbl001

Danielmeier, C., Wessel, J. R., Steinhauser, M., & Ullsperger, M. (2009). Modulation of the error-related negativity by response conflict. *Psychophysiology, 46*, 1288-1298. doi:10.1111/j.1469-8986.2009.00860.x

Denson, T. F., Spanovic, M., & Miller, N. (2009). Cognitive appraisals and emotions predict cortisol and immune responses: A meta-analysis of acute laboratory social stressors and emotion inductions. *Psychological Bulletin, 135*, 823-853. doi:10.1037/a0016909

Desimone, R., & Duncan, J. (1995). Neural mechanisms of selective visual attention. *Annual Review of Neuroscience, 18*, 193-222. doi:10.1146/annurev.ne.18.030195.001205

Dewall, C. N., Macdonald, G., Webster, G. D., Masten, C. L., Baumeister, R. F., Powell, C., . . . Eisenberger, N. I. (2010). Acetaminophen reduces social pain: Behavioral and neural evidence. *Psychological Science, 21*, 931-937. doi:10.1177/0956797610374741

DeWall, C. N., Masten, C. L., Powell, C., Combs, D., Schurtz, D. R., & Eisenberger, N. I. (2012). Do neural responses to rejection depend on attachment style? An fMRI study. *Social Cognitive and Affective Neuroscience, 7*, 184-192. doi:10.1093/scan/nsq107

Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: Shame, physiology, and health. *Journal of Personality, 72*, 1191-1216. doi:10.1111/j.1467-6494.2004.00295.x

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355-391. doi:10.1037/0033-2909.130.3.355

Dien, J. (2010). The ERP PCA Toolkit: An open source program for advanced statistical analysis of event-related potential data. *Journal of Neuroscience Methods, 187*, 138-145. doi:10.1016/j.jneumeth.2009.12.009

Dien, J. (2017). Best practices for repeated measures ANOVAs of ERP data: Reference, regional channels, and robust ANOVAs. *International Journal of Psychophysiology, 111*, 42-56. doi:10.1016/j.ijpsycho.2016.09.006

Dien, J., Franklin, M. S., & May, C. J. (2006). Is "Blank" a suitable neutral prime for event-related potential experiments? *Brain and Language, 97*, 91-101. doi:10.1016/j.bandl.2005.08.002

Dien, J., Michelson, C. A., & Franklin, M. S. (2010). Separating the visual sentence N400 effect from the P400 sequential expectancy effect: Cognitive and neuroanatomical implications. *Brain Research, 1355*, 126-140. doi:10.1016/j.brainres.2010.07.099

Dien, J., & Santuzzi, A. M. (2005). Principal components analysis of event-related potential datasets. In T. Handy (Ed.), *Event-Related Potentials: A Methods Handbook*. Cambridge, Massachusetts: MIT Press.

Dixon-Gordon, K. L., Gratz, K. L., Breetz, A., & Tull, M. (2013). A laboratory-based examination of responses to social rejection in borderline personality disorder: The mediating role of emotion dysregulation. *Journal of Personality Disorders, 27*, 157-171. doi:10.1521/pedi.2013.27.2.157

Docherty, N. M., St-Hilaire, A., Aakre, J. M., & Seghers, J. P. (2009). Life events and high-trait reactivity together predict psychotic symptom increases in schizophrenia. *Schizophrenia Bulletin, 35*, 638-645. doi:10.1093/schbul/sbn002

Donchin, E. (1981). Presidential address, 1980. Surprise!...Surprise? *Psychophysiology, 18*, 493-513. doi:10.1111/j.1469-8986.1981.tb01815.x

Donchin, E., & Coles, M. G. H. (1988). Is the P300 component a manifestation of context updating. *Behavioral and Brain Sciences, 11*, 355-372. doi:10.1017/S0140525X00058027

Duncan, J. (2010). The multiple-demand (MD) system of the primate brain: Mental programs for intelligent behaviour. *Trends in Cognitive Sciences, 14*, 172-179. doi:10.1016/j.tics.2010.01.004

Duncan-Johnson, C. C., & Donchin, E. (1977). On quantifying surprise: The variation of event-related potentials with subjective probability. *Psychophysiology, 14*, 456-467. doi:10.1111/j.1469-8986.1977.tb01312.x

Durston, S., Davidson, M. C., Thomas, K. M., Worden, M. S., Tottenham, N., Martinez, A., . . . Casey, B. J. (2003). Parametric manipulation of conflict and response competition using rapid mixed-trial event-related fMRI. *NeuroImage, 20*, 2135-2141. doi:10.1016/j.neuroimage.2003.08.004

Egner, T. (2011). Right ventrolateral prefrontal cortex mediates individual differences in conflict-driven cognitive control. *Journal of Cognitive Neuroscience, 23*, 3903-3913. doi:10.1162/jocn_a_00064

Egner, T., & Hirsch, J. (2005a). Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nature Neuroscience, 8*, 1784-1790. doi:10.1038/nn1594

Egner, T., & Hirsch, J. (2005b). The neural correlates and functional integration of cognitive control in a Stroop task. *NeuroImage, 24*, 539-547. doi:10.1016/j.neuroimage.2004.09.007

Eisenberger, N. I. (2012). The pain of social disconnection: Examining the shared neural underpinnings of physical and social pain. *Nature Reviews Neuroscience, 13*, 421-434. doi:10.1038/nrn3231

Eisenberger, N. I. (2013). Social ties and health: A social neuroscience perspective. *Current Opinion in Neurobiology, 23*, 407-413. doi:10.1016/j.conb.2013.01.006

Eisenberger, N. I. (2015). Social pain and the brain: Controversies, questions, and where to go from here. *Annual Review of Psychology, 66*, 601-629. doi:10.1146/annurev-psych-010213-115146

Eisenberger, N. I., & Cole, S. W. (2012). Social neuroscience and health: Neurophysiological mechanisms linking social ties with physical health. *Nature Neuroscience, 15*, 669-674. doi:10.1038/nn.3086

Eisenberger, N. I., Gable, S. L., & Lieberman, M. D. (2007). Functional magnetic resonance imaging responses relate to differences in real-world social experience. *Emotion 7*, 745-754. doi:10.1037/1528-3542.7.4.745

Eisenberger, N. I., Inagaki, T. K., Muscatell, K. A., Byrne Haltom, K. E., & Leary, M. R. (2011). The neural sociometer: Brain mechanisms underlying state self-esteem. *Journal of Cognitive Neuroscience, 23*, 3448-3455. doi:10.1162/jocn_a_00027

Eisenberger, N. I., Jarcho, J. M., Lieberman, M. D., & Naliboff, B. D. (2006). An experimental study of shared sensitivity to physical pain and social rejection. *Pain, 126*, 132-138. doi:10.1016/j.pain.2006.06.024

Eisenberger, N. I., & Lieberman, M. D. (2004). Why rejection hurts: A common neural alarm system for physical and social pain. *Trends in Cognitive Sciences, 8*, 294-300. doi:10.1016/j.tics.2004.05.010

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An FMRI study of social exclusion. *Science, 302*, 290-292. doi:10.1126/science.1089134

Eisenberger, N. I., Way, B. M., Taylor, S. E., Welch, W. T., & Lieberman, M. D. (2007). Understanding genetic risk for aggression: Clues from the brain's response to social exclusion. *Biological Psychiatry, 61*, 1100-1108. doi:10.1016/j.biopsych.2006.08.007

Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences, 15*, 85-93. doi:10.1016/j.tics.2010.11.004

Farrell, M. J., Laird, A. R., & Egan, G. F. (2005). Brain activity associated with painfully hot stimuli applied to the upper limb: A meta-analysis. *Human Brain Mapping, 25*, 129-139. doi:10.1002/hbm.20125

Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods, 41*, 1149-1160. doi:10.3758/BRM.41.4.1149

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-191. doi:10.3758/BF03193146

Folstein, J. R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. *Psychophysiology, 45*, 152-170. doi:10.1111/j.1469-8986.2007.00602.x

Foltz, E. L., & White Jr, L. E. (1962). Pain “relief” by frontal cingulumotomy. *Journal of Neurosurgery, 19*, 89-100.

Galashan, D., Fehr, T., & Herrmann, M. (2015). Differences between target and non-target probe processing — Combined evidence from fMRI, EEG and fMRI-constrained source analysis. *NeuroImage, 111*, 289-299. doi:10.1016/j.neuroimage.2015.02.044

Geniole, S. N., Carré, J. M., & McCormick, C. M. (2011). State, not trait, neuroendocrine function predicts costly reactive aggression in men after social exclusion and inclusion. *Biological Psychology, 87*, 137-145. doi:10.1016/j.biopsycho.2011.02.020

Gerlinger, G., Hauser, M., De Hert, M., Lacluyse, K., Wampers, M., & Correll, C. U. (2013). Personal stigma in schizophrenia spectrum disorders: A systematic review of prevalence rates, correlates, impact and interventions. *World Psychiatry, 12*, 155-164. doi:10.1002/wps.20040

Gonsalkorale, K., & Williams, K. D. (2007). The KKK won't let me play: Ostracism even by a despised outgroup hurts. *European Journal of Social Psychology, 37*, 1176-1186. doi:10.1002/ejsp.392

Gonzaga, G. C., Keltner, D., Londahl, E. A., & Smith, M. D. (2001). Love and the commitment problem in romantic relations and friendship. *Journal of Personality and Social Psychology, 81*, 247-262. doi:10.1037/0022-3514.81.2.247

Gonzalez, M. Z., Beckes, L., Chango, J., Allen, J. P., & Coan, J. A. (2015). Adolescent neighborhood quality predicts adult dACC response to social exclusion. *Social Cognitive and Affective Neuroscience, 10*, 921-928. doi:10.1093/scan/nsu137

Gradin, V. B., Waiter, G., Kumar, P., Stickle, C., Milders, M., Matthews, K., . . . Steele, J. D. (2012). Abnormal neural responses to social exclusion in schizophrenia. *PloS one, 7*, e42608. doi:10.1371/journal.pone.0042608.s004

Gündel, H., O'Connor, M.-F., Littrell, L., Fort, C., & Lane, R. D. (2003). Functional neuroanatomy of grief: An FMRI study. *The American Journal of Psychiatry, 160*, 1946-1953. doi:10.1176/appi.ajp.160.11.1946

Gutz, L., Küpper, C., Renneberg, B., & Niedeggen, M. (2011). Processing social participation: An event-related brain potential study. *NeuroReport, 22*, 453-458. doi:10.1097/WNR.0b013e3283476b67

Hadland, K. A., Rushworth, M. F. S., Gaffan, D., & Passingham, R. E. (2003). The effect of cingulate lesions on social behaviour and emotion. *Neuropsychologia, 41*, 919-931. doi:10.1016/S0028-3932(02)00325-1

Hajcak, G., McDonald, N., & Simons, R. F. (2004). Error-related psychophysiology and negative affect. *Brain and Cognition, 56*, 189-197. doi:10.1016/j.bandc.2003.11.001

Hammer, M. (1963). Influence of small social networks as factors on mental hospital admission. *Human Organization, 22*, 243-251. doi:10.17730/humo.22.4.u015583596800205

Hammer, M. (1981). Social supports, social networks, and schizophrenia. *Schizophrenia Bulletin, 7*, 45-57. doi:10.1093/schbul/7.1.45

Hartgerink, C. H. J., van Beest, I., Wicherts, J. M., & Williams, K. D. (2015). The ordinal effects of ostracism: A meta-analysis of 120 Cyberball studies. *PloS one, 10*, e0127002-0127024. doi:10.1371/journal.pone.0127002

Hartikainen, K. M., & Knight, R. T. (2003). Lateral and orbital prefrontal cortex contributions to attention. In J. Polich (Ed.), *Detection of Change: Event-Related Potential and fMRI Findings* (pp. 99-116). Boston, MA: Kluwer Academic Press.

Hoffman, R. E. (2007). A social deafferentation hypothesis for induction of active schizophrenia. *Schizophrenia Bulletin, 33*, 1066-1070. doi:10.1093/schbul/sbm079

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*, 679-709. doi:10.1037/0033-295x.109.4.679

Holroyd, C. B., & Coles, M. G. H. (2008). Dorsal anterior cingulate cortex integrates reinforcement history to guide voluntary behavior. *Cortex, 44*, 548-559. doi:10.1016/j.cortex.2007.08.013

Holroyd, C. B., Yeung, N., Coles, M. G., & Cohen, J. D. (2005). A mechanism for error detection in speeded response time tasks. *Journal of Experimental Psychology: General, 134*, 163-191. doi:10.1037/0096-3445.134.2.163

Hooley, J. M. (2007). Expressed emotion and relapse of psychopathology. *Annual Review of Clinical Psychology, 3*, 329-352. doi:10.1146/annurev.clinpsy.2.022305.095236

Hopstaken, J. F., van der Linden, D., Bakker, A. B., & Kompier, M. A. J. (2015). A multifaceted investigation of the link between mental fatigue and task disengagement. *Psychophysiology, 52*, 305-315. doi:10.1111/psyp.12339

Horan, W. P., Ventura, J., Nuechterlein, K. H., Subotnik, K. L., Hwang, S. S., & Mintz, J. (2005). Stressful life events in recent-onset schizophrenia: Reduced frequencies and altered subjective appraisals. *Schizophrenia Research, 75*, 363-374. doi:10.1016/j.schres.2004.07.019

Howard, L., Leese, M., & Thornicroft, G. (2000). Social networks and functional status in patients with psychosis. *Acta Psychiatrica Scandinavica, 102*, 376-385. doi:10.1034/j.1600-0447.2000.102005376.x

Huotilainen, M., Ilmoniemi, R. J., Tiitinen, H., Lavikainen, J., Alho, K., Kajola, M., & Näätänen, R. (1995). The projection method in removing eye-blink artefacts from multichannel MEG measurements. In C. Baumgartner, L. Deecke, G. Stroink, & S. J. Williamson (Eds.), *Biomagnetism: Fundamental Research and Clinical Applications* (pp. 363-367): Elsevier Science, IOS Press.

Ibarra-Rovillard, M. S., & Kuiper, N. A. (2011). Social support and social negativity findings in depression: Perceived responsiveness to basic psychological needs. *Clinical Psychology Review, 31*, 342-352. doi:10.1016/j.cpr.2011.01.005

Ille, N., Berg, P., & Scherg, M. (2002). Artifact correction of the ongoing EEG using spatial filters based on artifact and brain signal topographies. *Journal of Clinical Neurophysiology, 19*, 113-124.

Kalin, N. H., Shelton, S. E., Fox, A. S., Oakes, T. R., & Davidson, R. J. (2005). Brain regions associated with the expression and contextual regulation of anxiety in primates. *Biological Psychiatry, 58*, 796-804. doi:10.1016/j.biopsych.2005.05.021

Kawamoto, T., Onoda, K., Nakashima, K., Nittono, H., Yamaguchi, S., & Ura, M. (2012). Is dorsal anterior cingulate cortex activation in response to social exclusion due to expectancy violation? An fMRI study. *Frontiers in Evolutionary Neuroscience, 4*, 11. doi:10.3389/fnevo.2012.00011

Kelly, B. D., O'Callaghan, E., Waddington, J. L., Feeney, L., Browne, S., Scully, P. J., . . . Larkin, C. (2010). Schizophrenia and the city: A review of literature and prospective study of psychosis and urbanicity in Ireland. *Schizophrenia Research, 116*, 75-89. doi:10.1016/j.schres.2009.10.015

Kemeny, M. E., Gruenewald, T. L., & Dickerson, S. S. (2004). Shame as the emotional response to threat to the social self: Implications for behavior, physiology, and health. *Psychological Inquiry, 15*, 153-160. doi:10.1016/j.bbi.2005.11.005

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science, 303*, 1023-1026. doi:10.1126/science.1089910

Kersting, A., Ohrmann, P., Pedersen, A., Kroker, K., Samberg, D., Bauer, J., . . . Suslow, T. (2009). Neural activation underlying acute grief in women after the loss of an unborn child. *The American Journal of Psychiatry, 166*, 1402-1410. doi:10.1176/appi.ajp.2009.08121875

Keselman, H. J., Wilcox, R. R., & Lix, L. M. (2003). A generally robust approach to hypothesis testing in independent and correlated groups designs. *Psychophysiology, 40*, 586-596. doi:10.1111/1469-8986.00060

Khosla, D., Don, M., & Kwong, B. (1999). Spatial mislocalization of EEG electrodes – Effects on accuracy of dipole estimation. *Clinical Neurophysiology, 110*, 261-271. doi:10.1016/S0013-4694(98)00121-7

Kiehl, K. A., Laurens, K. R., Duty, T. L., Forster, B. B., & Liddle, P. F. (2001). Neural sources involved in auditory target detection and novelty processing: An event-related fMRI study. *Psychophysiology, 38*, 133-142. doi:10.1111/1469-8986.3810133

Kirkbride, J. B., Errazuriz, A., Croudace, T. J., Morgan, C., Jackson, D., Boydell, J., . . . Jones, P. B. (2012). Incidence of schizophrenia and other psychoses in England, 1950-2009: A systematic review and meta-analyses. *PloS one, 7*, e31660. doi:10.1371/journal.pone.0031660

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76-81. doi:10.1159/000119004

Koessler, L., Maillard, L., Benhadid, A., Vignal, J. P., Braun, M., & Vespignani, H. (2007). Spatial localization of EEG electrodes. *Clinical Neurophysiology, 37*, 97-102. doi:10.1016/j.neucli.2007.03.002

Koivumaa-Honkanen, H. T., Honkanen, R., Antikainen, R., Hintikka, J., & Viinamäki, H. (1999). Self-reported life satisfaction and treatment factors in patients with schizophrenia, major depression and anxiety disorder. *Acta Psychiatrica Scandinavica, 99*, 377-384. doi:10.1111/j.1600-0447.1999.tb07244.x

Kok, A. (2001). On the utility of P3 amplitude as a measure of processing capacity. *Psychophysiology, 38*, 557-577. doi:10.1017/S0048577201990559

Krabbendam, L., & van Os, J. (2005). Schizophrenia and urbanicity: A major environmental influence--conditional on genetic risk. *Schizophrenia Bulletin, 31*, 795-799. doi:10.1093/schbul/sbi060

Kross, E., Egner, T., Ochsner, K., Hirsch, J., & Downey, G. (2007). Neural dynamics of rejection sensitivity. *Journal of Cognitive Neuroscience, 19*, 945-956. doi:10.1162/jocn.2007.19.6.945

Kuipers, E., Bebbington, P., Dunn, G., Fowler, D., Freeman, D., Watson, P., . . . Garety, P. (2006). Influence of carer expressed emotion and affect on relapse in non-affective psychosis. *The British Journal of Psychiatry, 188*, 173-179. doi:10.1192/bjp.bp.104.007294

Kulkarni, B., Bentley, D. E., Elliott, R., Youell, P., Watson, A., Derbyshire, S. W. G., . . . Jones, A. K. P. (2005). Attention to pain localization and unpleasantness discriminates the functions of the medial and lateral pain systems. *The European Journal of Neuroscience, 21*, 3133-3142. doi:10.1111/j.1460-9568.2005.04098.x

Ladouceur, C. D., Dahl, R. E., & Carter, C. S. (2007). Development of action monitoring through adolescence into adulthood: ERP and source localization. *Developmental Science, 10*, 874-891. doi:10.1111/j.1467-7687.2007.00639.x

Lamm, C., & Singer, T. (2010). The role of anterior insular cortex in social emotions. *Brain Structure & Function, 214*, 579-591. doi:10.1007/s00429-010-0251-3

Larson, M. J., Clawson, A., Clayson, P. E., & Baldwin, S. A. (2013). Cognitive conflict adaptation in generalized anxiety disorder. *Biological Psychology, 94*, 408-418. doi:10.1016/j.biopsycho.2013.08.006

Larson, M. J., Clayson, P. E., & Clawson, A. (2014). Making sense of all the conflict: A theoretical review and critique of conflict-related ERPs. *International Journal of Psychophysiology, 93*, 283-297. doi:10.1016/j.ijpsycho.2014.06.007

Lataster, J., Myin-Germeys, I., Lieb, R., Wittchen, H. U., & Van Os, J. (2012). Adversity and psychosis: A 10-year prospective study investigating synergism between early and recent adversity in psychosis. *Acta Psychiatrica Scandinavica, 125*, 388-399. doi:10.1111/j.1600-0447.2011.01805.x

Lebell, M. B., Marder, S. R., Mintz, J., Mintz, L. I., Tompson, M., Wirshing, W., . . . McKenzie, J. (1993). Patients' perceptions of family emotional climate and outcome in schizophrenia. *The British Journal of Psychiatry 162*, 751-754. doi:10.1192/bjp.162.6.751

Livingston, J. D., & Boyd, J. E. (2010). Correlates and consequences of internalized stigma for people living with mental illness: A systematic review and meta-analysis. *Social Science & Medicine, 71*, 2150-2161. doi:10.1016/j.socscimed.2010.09.030

Luck, S. J. (2014). *An introduction to the event-related potential technique* (2nd ed.). Cambridge, MA: The MIT Press.

Luu, P., Collins, P., & Tucker, D. M. (2000). Mood, personality, and self-monitoring: Negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring.

Journal of Experimental Psychology: General, 129, 43-60. doi:10.1037/0096-3445.129.1.43

Macdonald, G., & Leary, M. R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin, 131*, 202-223. doi:10.1037/0033-2909.131.2.202

MacLean, P. D., & Newman, J. D. (1988). Role of midline frontolimbic cortex in production of the isolation call of squirrel monkeys. *Brain Research, 450*, 111-123. doi:10.1016/0006-8993(88)91550-8

Major, B., & O'Brien, L. T. (2005). The social psychology of stigma. *Annual Review of Psychology, 56*, 393-421. doi:10.1146/annurev.psych.56.091103.070137

March, D., Hatch, S. L., Morgan, C., Kirkbride, J. B., Bresnahan, M., Fearon, P., & Susser, E. (2008). Psychosis and place. *Epidemiologic Reviews, 30*, 84-100. doi:10.1093/epirev/mxn006

Masten, C. L., Eisenberger, N. I., Borofsky, L. A., Pfeifer, J. H., McNealy, K., Mazziotta, J. C., & Dapretto, M. (2009). Neural correlates of social exclusion during adolescence: Understanding the distress of peer rejection. *Social Cognitive and Affective Neuroscience, 4*, 143-157. doi:10.1093/scan/nsp007

Masten, C. L., Telzer, E. H., & Eisenberger, N. I. (2011). An FMRI investigation of attributing negative social treatment to racial discrimination. *Journal of Cognitive Neuroscience, 23*, 1042-1051. doi:10.1162/jocn.2010.21520

Masten, C. L., Telzer, E. H., Fuligni, A. J., Lieberman, M. D., & Eisenberger, N. I. (2012). Time spent with friends in adolescence relates to less neural sensitivity to later peer rejection. *Social Cognitive and Affective Neuroscience, 7*, 106-114. doi:10.1093/scan/nsq098

McPartland, J. C., Crowley, M. J., Perszyk, D. R., Naples, A. J., Mukerji, C. E., Wu, J., . . . Mayes, L. C. (2011). Temporal dynamics reveal atypical brain response to social exclusion in autism. *Developmental Cognitive Neuroscience, 1*, 271-279. doi:10.1016/j.dcn.2011.02.003

Mechias, M.-L., Etkin, A., & Kalisch, R. (2010). A meta-analysis of instructed fear studies: Implications for conscious appraisal of threat. *NeuroImage, 49*, 1760-1768. doi:10.1016/j.neuroimage.2009.09.040

Menon, V., Adleman, N. E., White, C. D., Glover, G. H., & Reiss, A. L. (2001). Error-related brain activation during a Go/NoGo response inhibition task. *Human Brain Mapping, 12*, 131-143. doi:10.1002/1097-0193(200103)12:3<131::AID-HBM1010>3.0.CO;2-C

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28*, 487-495. doi:10.1016/0005-7967(90)90135-6

Meyer-Lindenberg, A., & Tost, H. (2012). Neural mechanisms of social risk for psychiatric disorders. *Nature Neuroscience, 15*, 663-668. doi:10.1038/nn.3083

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501-524. doi:10.1146/annurev.psych.60.110707.163551

Mobbs, D., Marchant, J. L., Hassabis, D., Seymour, B., Tan, G., Gray, M., . . . Frith, C. D. (2009). From threat to fear: The neural organization of defensive fear systems in humans. *The Journal of Neuroscience 29*, 12236-12243. doi:10.1523/JNEUROSCI.2378-09.2009

Mobbs, D., Petrovic, P., Marchant, J. L., Hassabis, D., Weiskopf, N., Seymour, B., . . . Frith, C. D. (2007). When fear is near: Threat imminence elicits prefrontal-periaqueductal gray shifts in humans. *Science, 317*, 1079-1083. doi:10.1126/science.1144298

Mondelli, V., Dazzan, P., Gabilondo, A., Tournikioti, K., Walshe, M., Marshall, N., . . . Pariante, C. M. (2008). Pituitary volume in unaffected relatives of patients with schizophrenia and bipolar disorder. *Psychoneuroendocrinology, 33*, 1004-1012. doi:10.1016/j.psyneuen.2008.05.010

Morgan, C., Charalambides, M., Hutchinson, G., & Murray, R. M. (2010). Migration, ethnicity, and psychosis: Toward a sociodevelopmental model. *Schizophrenia Bulletin, 36*, 655-664. doi:10.1093/schbul/sbq051

Morgan, C., Kirkbride, J., Hutchinson, G., Craig, T., Morgan, K., Dazzan, P., . . . Fearon, P. (2008). Cumulative social disadvantage, ethnicity and first-episode psychosis: A case-control study. *Psychological Medicine, 38*, 1701-1715. doi:10.1017/S0033291708004534

Murphy, M. R., MacLean, P. D., & Hamilton, S. C. (1981). Species-typical behavior of hamsters deprived from birth of the neocortex. *Science, 213*, 459-461. doi:10.1126/science.7244642

Murphy, P. R., Robertson, I. H., Balsters, J. H., & O'Connell, R. G. (2011). Pupillometry and P3 index the locus coeruleus-noradrenergic arousal function in humans. *Psychophysiology, 48*, 1532-1543. doi:10.1111/j.1469-8986.2011.01226.x

Myin-Germeys, I., Van Os, J., Schwartz, J. E., Stone, A. A., & Delespaul, P. A. (2001). Emotional reactivity to daily life stress in psychosis. *Archives of General Psychiatry, 58*, 1137-1144. doi:10.1001/archpsyc.58.12.1137

Nachev, P., Wydell, H., Neill, K. O., Husain, M., & Kennard, C. (2007). The role of the pre-supplementary motor area in the control of action. *NeuroImage, 36*, T155-T163. doi:10.1016/j.neuroimage.2007.03.034

Niedeggen, M., Sarauli, N., Cacciola, S., & Weschke, S. (2014). Are there benefits of social overinclusion? Behavioral and ERP effects in the Cyberball paradigm. *Frontiers in Human Neuroscience, 8*, 935. doi:10.3389/fnhum.2014.00935

Norman, R. M., & Malla, A. K. (1991). Subjective stress in schizophrenic patients. *Social Psychiatry and Psychiatric Epidemiology, 26*, 212-216. doi:10.1007/BF00788968

Norman, R. M., & Malla, A. K. (1993). Stressful life events and schizophrenia. I: A review of the research. *The British Journal of Psychiatry, 162*, 161-166.

Norman, R. M., Malla, A. K., Manchanda, R., Harricharan, R., Takhar, J., & Northcott, S. (2005). Social support and three-year symptom and admission outcomes for first episode psychosis. *Schizophrenia Research, 80*, 227-234. doi:10.1016/j.schres.2005.05.006

Nuechterlein, K. H., & Dawson, M. E. (1984). A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin, 10*, 300-312. doi:10.1093/schbul/10.2.300

Nuechterlein, K. H., Dawson, M. E., Ventura, J., Gitlin, M., Subotnik, K. L., Snyder, K. S., . . . Bartzokis, G. (1994). The vulnerability/stress model of schizophrenic relapse: A longitudinal study. *Acta Psychiatrica Scandinavica, 382*, 58-64. doi:10.1111/j.1600-0447.1994.tb05867.x

O'Connor, M.-F., Wellisch, D. K., Stanton, A. L., Eisenberger, N. I., Irwin, M. R., & Lieberman, M. D. (2008). Craving love? Enduring grief activates brain's reward center. *NeuroImage, 42*, 969-972. doi:10.1016/j.neuroimage.2008.04.256

Olvet, D. M., & Hajcak, G. (2009). The effect of trial-to-trial feedback on the error-related negativity and its relationship with anxiety. *Cognitive Affective and Behavioral Neuroscience, 9*, 427-433. doi:10.3758/CABN.9.4.427

Olvet, D. M., & Hajcak, G. (2012). The error-related negativity relates to sadness following mood induction among individuals with high neuroticism. *Social Cognitive and Affective Neuroscience, 7*, 289-295. doi:10.1093/scan/nsr007

Onoda, K., Okamoto, Y., Nakashima, K., Nittono, H., Ura, M., & Yamawaki, S. (2009). Decreased ventral anterior cingulate cortex activity is associated with reduced social pain during emotional support. *Social Neuroscience, 4*, 443-454. doi:10.1080/17470910902955884

Onoda, K., Okamoto, Y., Nakashima, K., Nittono, H., Yoshimura, S., Yamawaki, S., . . . Ura, M. (2010). Does low self-esteem enhance social pain? The relationship between trait self-esteem and anterior cingulate cortex activation induced by ostracism. *Social Cognitive and Affective Neuroscience, 5*, 385-391.

Oostenveld, R., & Praamstra, P. (2001). The five percent electrode system for high-resolution EEG and ERP measurements. *Clinical Neurophysiology, 112*, 713-719. doi:10.1016/S1388-2457(00)00527-7

Patterson, T. L., Semple, S. J., Shaw, W. S., Halpain, M., Moscona, S., Grant, I., & Jeste, D. V. (1997). Self-reported social functioning among older patients with schizophrenia. *Schizophrenia Research, 27*, 199-210. doi:10.1016/S0920-9964(97)00078-9

Paul-Jordanov, I., Hoechstetter, K., Bornfleth, H., Waelkens, A., & Scherg, M. (2016). *BESA Research 6.1 User Manual*. Graefelfing, Germany: BESA GmbH.

Paus, T., Koski, L., Caramanos, Z., & Westbury, C. (1998). Regional differences in the effects of task difficulty and motor output on blood flow response in the human anterior cingulate cortex: A review of 107 PET activation studies. *NeuroReport, 9*, R37-47. doi:10.1097/00001756-199806220-00001

Pedersen, C. B., & Mortensen, P. B. (2001). Evidence of a dose-response relationship between urbanicity during upbringing and schizophrenia risk. *Archives of General Psychiatry, 58*, 1039-1046. doi:10.1001/archpsyc.58.11.1039

Pessoa, L. (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Sciences, 13*, 160-166. doi:10.1016/j.tics.2009.01.006

Peyron, R., Laurent, B., & García-Larrea, L. (2000). Functional imaging of brain responses to pain. A review and meta-analysis (2000). *Clinical Neurophysiology, 30*, 263-288. doi:10.1016/S0987-7053(00)00227-6

Phan, K. L., Wager, T. D., Taylor, S. F., & Liberzon, I. (2004). Functional neuroimaging studies of human emotions. *CNS Spectrums, 9*, 258-266. doi:10.1017/S1092852900009196

Phillips, L. J., Francey, S. M., Edwards, J., & McMurray, N. (2007). Stress and psychosis: Towards the development of new models of investigation. *Clinical Psychology Review, 27*, 307-317. doi:10.1016/j.cpr.2006.10.003

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology, 118*, 2128-2148. doi:10.1016/j.clinph.2007.04.019

Polich, J. (2012). Neuropsychology of P300. In S. J. Luck & E. S. Kappenman (Eds.), *Oxford Handbook of Event-Related Potential Components* (pp. 159-188). New York, NY: Oxford University Press, Inc.

Price, D. D. (2000). Psychological and neural mechanisms of the affective dimension of pain. *Science, 288*, 1769-1772. doi:10.1126/science.288.5472.1769

Puetz, V. B., Kohn, N., Dahmen, B., Zvyagintsev, M., Schüppen, A., Schultz, R. T., . . . Konrad, K. (2014). Neural response to social rejection in children with early separation experiences. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*, 1328-1337. e1328.

Rainville, P. (2002). Brain mechanisms of pain affect and pain modulation. *Current Opinion in Neurobiology, 12*, 195-204. doi:10.1016/S0959-4388(02)00313-6

Renwick, L., Jackson, D., Turner, N., Sutton, M., Foley, S., McWilliams, S., . . . O'Callaghan, E. (2009). Are symptoms associated with increased levels of perceived stress in first-episode psychosis? *International Journal of Mental Health Nursing, 18*, 186-194. doi:10.1111/j.1447-0349.2009.00600.x

Rohleder, N. (2014). Stimulation of systemic low-grade inflammation by psychosocial stress. *Psychosomatic Medicine, 76*, 181-189. doi:10.1097/PSY.0000000000000049

Rotge, J. Y., Lemogne, C., Hinfray, S., Huguet, P., Grynszpan, O., Tartour, E., . . . Fossati, P. (2014). A meta-analysis of the anterior cingulate contribution to social pain. *Social Cognitive and Affective Neuroscience, 10*, 19-27. doi:10.1093/scan/nsu110

Rubia, K., Russell, T., Overmeyer, S., Brammer, M. J., Bullmore, E. T., Sharma, T., . . . Taylor, E. (2001). Mapping motor inhibition: Conjunctive brain activations across different versions of Go/No-Go and stop tasks. *NeuroImage, 13*, 250-261. doi:10.1006/nimg.2000.0685

Rudebeck, P. H., Buckley, M. J., Walton, M. E., & Rushworth, M. F. S. (2006). A role for the macaque anterior cingulate gyrus in social valuation. *Science, 313*, 1310-1312. doi:10.1126/science.1128197

Rüsch, N., Müller, M., Lay, B., Corrigan, P. W., Zahn, R., Schönenberger, T., . . . Rössler, W. (2014). Emotional reactions to involuntary psychiatric hospitalization and stigma-related stress among people with mental illness. *European Archives of Psychiatry and Clinical Neuroscience, 264*, 35-43. doi:10.1007/s00406-013-0412-5

Russell, J. A., & Carroll, J. M. (1999). On the bipolarity of positive and negative affect. *Psychological Bulletin, 125*, 3-30. doi:10.1037/0033-2909.125.1.3

Sarason, I. G., Levine, H. M., Basham, R. B., & Sarason, B. R. (1983). Assessing social support: The social support questionnaire. *Journal of Personality and Social Psychology, 44*, 127. doi:10.1037/0022-3514.44.1.127

Scherg, M., & Berg, P. (1996). New concepts of brain source imaging and localization. *Electroencephalography and Clinical Neurophysiology Supplement, 46*, 127-137.

Scherg, M., Ille, N., Bornfleth, H., & Berg, P. (2002). Advanced tools for digital EEG review: Virtual source montages, whole-head mapping, correlation, and phase analysis. *Journal of Clinical Neurophysiology, 19*, 91-112.

Scherg, M., & Von Cramon, D. (1986). Evoked dipole source potentials of the human auditory cortex. *Electroencephalography and Clinical Neurophysiology, 65*, 344-360.

Schiller, D., Levy, I., Niv, Y., LeDoux, J. E., & Phelps, E. A. (2008). From fear to safety and back: Reversal of fear in the human brain. *The Journal of Neuroscience 28*, 11517-11525. doi:10.1523/JNEUROSCI.2265-08.2008

Seeman, T. E. (1996). Social ties and health: The benefits of social integration. *Annals of Epidemiology, 6*, 442-451. doi:10.1016/S1047-2797(96)00095-6

Seidel, E. M., Silani, G., Metzler, H., Thaler, H., Lamm, C., Gur, R. C., . . . Derntl, B. (2013). The impact of social exclusion vs. inclusion on subjective and hormonal reactions in females and males. *Psychoneuroendocrinology, 38*, 2925-2932. doi:10.1016/j.psyneuen.2013.07.021

Selten, J.-P., & Cantor-Graae, E. (2005). Social defeat: Risk factor for schizophrenia? *The British journal of Psychiatry, 187*, 101-102. doi:10.1192/bjp.187.2.101

Selten, J.-P., Cantor-Graae, E., & Kahn, R. S. (2007). Migration and schizophrenia. *Current Opinion in Psychiatry, 20*, 111-115. doi:10.1097/YCO.0b013e328017f68e

Selten, J.-P., van der Ven, E., Rutten, B. P. F., & Cantor-Graae, E. (2013). The social defeat hypothesis of schizophrenia: An update. *Schizophrenia Bulletin, 39*, 1180-1186. doi:10.1093/schbul/sbt134

Semple, S. J., Patterson, T. L., Shaw, W. S., Grant, I., Moscona, S., Koch, W., & Jeste, D. (1997). The social networks of older schizophrenia patients. *International Psychogeriatrics, 9*, 81-94. doi:10.1017/S1041610297004237

Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nature Reviews Neuroscience, 12*, 154-167. doi:10.1038/nrn2994

Sholty, G. L. (2012). Psychobiological determinants of stress reactivity in schizophrenia (Doctoral Dissertation). Retrieved from http://escholarship.org/uc/item/50g1d5p2.

Silton, R. L., Heller, W., Towers, D. N., Engels, A. S., Spielberg, J. M., Edgar, J. C., . . . Miller, G. A. (2010). The time course of activity in dorsolateral prefrontal cortex and anterior cingulate cortex during top-down attentional control. *NeuroImage, 50*, 1292-1302. doi:10.1016/j.neuroimage.2009.12.061

Simons, R. F., Graham, F. K., Miles, M. A., & Chen, X. (2001). On the relationship of P3a and the Novelty-P3. *Biological Psychology, 56*, 207-218. doi:On the relationship of P3a and the Novelty-P3.

Slavich, G. M., Way, B. M., Eisenberger, N. I., & Taylor, S. E. (2010). Neural sensitivity to social rejection is associated with inflammatory responses to social stress. *Proceedings of the National Academy of Sciences of the United States of America, 107*, 14817-14822. doi:10.1073/pnas.1009164107

Smith, E. R., Murphy, J., & Coats, S. (1999). Attachment to groups: Theory and measurement. *Journal of Personality and Social Psychology, 77*, 94-110. doi:10.1037/0022-3514.77.1.94

Sokolovsky, J., Cohen, C., Berger, D., & Geiger, J. (1978). Personal networks of ex-mental patients in a Manhattan SRO hotel. *Human Organization, 37*, 5-15. doi:10.17730/humo.37.1.m7015m7717632840

Spunt, R. P., Lieberman, M. D., Cohen, J. R., & Eisenberger, N. I. (2012). The phenomenology of error processing: The dorsal ACC response to stop-signal errors tracks reports of negative affect. *Journal of Cognitive Neuroscience, 24*, 1753-1765. doi:10.1162/jocn_a_00242

Squires, K. C., Wickens, C., Squires, N. K., & Donchin, E. (1976). The effect of stimulus sequence on the waveform of the cortical event-related potential. *Science, 193*, 1142-1146. doi:10.1111/j.1469-8986.1977.tb01312.x

Stan Development Team. (2017). CmdStan: The command-line interface to Stan, version 2.14.0. Retrieved from http://mc-stan.org/

Steiger, J. H. (1980a). Testing pattern hypotheses on correlation matrices: Alternative statistics and some empirical results. *Multivariate Behavioral Research, 15*, 335-352. doi:10.1207/s15327906mbr1503_7

Steiger, J. H. (1980b). Tests for comparing elements of a correlation matrix. *Psychological Bulletin, 87*, 245-251.

Stemmer, B., Segalowitz, S. J., Witzke, W., & Schonle, P. W. (2004). Error detection in patients with lesions to the medial prefrontal cortex: An ERP study. *Neuropsychologia, 42*, 118-130. doi:10.1016/S0028-3932(03)00121-0

Świtaj, P., Wciórka, J., Smolarska-Świtaj, J., & Grygiel, P. (2009). Extent and predictors of stigma experienced by patients with schizophrenia. *European Psychiatry 24*, 513-520. doi:10.1016/j.eurpsy.2009.06.003

Themanson, J. R., Ball, A. B., Khatcherian, S. M., & Rosen, P. J. (2014). The effects of social exclusion on the ERN and the cognitive control of action monitoring. *Psychophysiology, 51*, 215-225. doi:10.1111/psyp.12172

Themanson, J. R., Khatcherian, S. M., Ball, A. B., & Rosen, P. J. (2013). An event-related examination of neural activity during social interactions. *Social Cognitive and Affective Neuroscience, 8*, 727-733. doi:10.1093/scan/nss058

Themanson, J. R., Schreiber, J. A., Larsen, A. D., Dunn, K. R., Ball, A. B., & Khatcherian, S. M (2015). The ongoing cognitive processing of exclusionary social events: Evidence from event-related potentials. *Social Neuroscience, 10*, 55-69. doi:10.1080/17470919.2014.956899

Thornhill, R., & Thornhill, N. W. (1989). The evolution of psychological pain. In R. Bell (Ed.), *Sociobiology and the social sciences* (pp. 73-103). Lubbock, TX: Texas Tech University Press.

Tolle, T. R., Kaufmann, T., Siessmeier, T., Lautenbacher, S., Berthele, A., Munz, F., . . . Bartenstein, P. (2001). Region-specific encoding of sensory and affective components of pain in the human brain: A positron emission tomography correlation analysis. *Annals of Neurology, 45*, 40-47. doi:10.1002/1531-8249(199901)45:1<40::AID-ART8>3.0.CO;2-L

Tolsdorf, C. C. (1976). Social networks, support, and coping: An exploratory study. *Family Process, 15*, 407-417. doi:10.1111/j.1545-5300.1976.00407.x

Tompson, M. C., Goldstein, M. J., Lebell, M. B., Mintz, L. I., Marder, S. R., & Mintz, J. (1995). Schizophrenic patients' perceptions of their relatives' attitudes. *Psychiatry Research, 57*, 155-167. doi:10.1016/0165-1781(95)02598-Q

Treede, R.-D., Kenshalo, D. R., Gracely, R. H., & Jones, A. K. P. (1999). The cortical representation of pain. *Pain, 79*, 105-111. doi:10.1016/S0304-3959(98)00184-5

van Beest, I., & Williams, K. D. (2006). When inclusion costs and ostracism pays, ostracism still hurts. *Journal of Personality and Social Psychology, 91*, 918-928. doi:10.1037/0022-3514.91.5.918

van Os, J., Kenis, G., & Rutten, B. P. F. (2010). The environment and schizophrenia. *Nature, 468*, 203-212. doi:10.1038/nature09563

van Veen, V., & Carter, C. S. (2002). The anterior cingulate as a conflict monitor: fMRI and ERP studies. *Physiology & Behavior, 77*, 477-482. doi:10.1016/S0031-9384(02)00930-7

Vassos, E., Pedersen, C. B., Murray, R. M., Collier, D. A., & Lewis, C. M. (2012). Meta-analysis of the association of urbanicity with schizophrenia. *Schizophrenia Bulletin, 38*, 1118-1123. doi:10.1093/schbul/sbs096

Veling, W., Susser, E., van Os, J., Mackenbach, J. P., Selten, J.-P., & Hoek, H. W. (2008). Ethnic density of neighborhoods and incidence of psychotic disorders among immigrants. *The American Journal of Psychiatry, 165*, 66-73. doi:10.1176/appi.ajp.2007.07030423

Venkatraman, V., & Huettel, S. A. (2012). Strategic control in decision-making under uncertainty. *European Journal of Neuroscience, 35*, 1075-1082. doi:10.1111/j.1460-9568.2012.08009.x

Ventura, J., Nuechterlein, K. H., Lukoff, D., & Hardesty, J. P. (1989). A prospective study of stressful life events and schizophrenic relapse. *Journal of Abnormal Psychology, 98*, 407-411.

Vogt, B. A. (2005). Pain and emotion interactions in subregions of the cingulate gyrus. *Nature Reviews Neuroscience, 6*, 533-544. doi:10.1038/nrn1704

Walker, E., Mittal, V., & Tessner, K. (2008). Stress and the hypothalamic pituitary adrenal axis in the developmental course of schizophrenia. *Annual Review of Clinical Psychology, 4*, 189-216. doi:10.1146/annurev.clinpsy.4.022007.141248

Wang, Y., & Gotman, J. (2001). The influence of electrode location errors on EEG dipole source localization with a realistic head model. *Clinical Neurophysiology, 112*, 1777-1780. doi:10.1016/S1388-2457(01)00594-6

Watson, D., & Clark, L. A. (1993). Behavioral disinhibition versus constraint: A dispositional perspective. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of Mental Control* (pp. 506-527). New York, NY: Prentice Hall.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070. doi:10.1037/0022-3514.54.6.1063

Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104*, 15-25. doi:10.1037/0021-843X.104.1.15

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological bulletin, 98*, 219-235. doi:10.1037/0033-2909.98.2.219

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*, 3-14. doi:10.1037/0021-843X.104.1.3

Weinberg, A., Klein, D. N., & Hajcak, G. (2012). Increased error-related brain activity distinguishes generalized anxiety disorder with and without comorbid major depressive disorder. *Journal of Abnormal Psychology, 121*, 885-896. doi:10.1037/a0028270

Williams, K. D., Cheung, C. K., & Choi, W. (2000). Cyberostracism: Effects of being ignored over the Internet. *Journal of Personality and Social Psychology, 79*, 748-762. doi:10.1037/0022-3514.79.5.748

Williams, K. D., & Sommer, K. L. (1997). Social ostracism by coworkers: Does rejection lead to loafing or compensation? *Personality and Social Psychology Bulletin, 23*, 693-706. doi:10.1177/0146167297237003

Wiswede, D., Münte, T. F., & Rüsseler, J. (2009). Negative affect induced by derogatory verbal feedback modulates the neural signature of error detection. *Social Cognitive and Affective Neuroscience, 4*, 227-237. doi:10.1093/scan/nsp015

Xu, M., Li, Z., Diao, L., Zhang, L., Yuan, J., Ding, C., & Yang, D. (2016). Social exclusion modulates priorities of attention allocation in cognitive control. *Scientific Reports, 6*, 31282. doi:10.1038/srep31282

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*, 931-959. doi:10.1037/0033-295x.111.4.931

Zadro, L., Williams, K. D., & Richardson, R. (2004). How low can you go? Ostracism by a computer is sufficient to lower self-reported levels of belonging, control, self-esteem, and meaningful existence. *Journal of Experimental Social Psychology, 40*, 560-567. doi:10.1016/j.jesp.2003.11.006

Zhang, Q., Li, X., Wang, K., Zhou, X., Dong, Y., Zhang, L., . . . Yu, F. (2017). Dull to social acceptance rather than sensitivity to social ostracism in interpersonal interaction for depression: Behavioral and electrophysiological evidence from Cyberball tasks. *Frontiers in Human Neuroscience, 11*, 497-410. doi:10.3389/fnhum.2017.00162

Zöller, C., Maroof, P., Weik, U., & Deinzer, R. (2010). No effect of social exclusion on salivary cortisol secretion in women in a randomized controlled study. *Psychoneuroendocrinology, 35*, 1294-1298. doi:10.1016/j.psyneuen.2010.02.019

Zwolinski, J. (2012). Psychological and neuroendocrine reactivity to ostracism. *Aggressive Behavior, 38*, 108-125. doi:10.1002/ab.21411